CARDIAC COMPLICATIONS IN ACUTE ISCHEMIC/THROMBOTIC STROKE PATIENTS WITH SPECIAL REFERENCE TO VENTRICULAR DYSFUNCTION

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CERTIFICATE

This is to certify that the dissertation titled **"CARDIAC COMPLICATIONS IN ACUTE ISCHEMIC/THROMBOTIC STROKE PATIENTS WITH SPECIAL REFERENCE TO VENTRICULAR DYSFUNCTION''** is a bonafide work done by **Dr. VIVEK.M**, Post Graduate Student, Institute of Internal Medicine, Madras Medical College, Chennai-3, during March 2014 to August 2014 in partial fulfillment of the University requirements for the award of MD Branch-I General Medicine, under our guidance and supervision, during the academic year 2012-2015.

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DECLARATION

I, **Dr.VIVEK.M** solemnly declare that dissertation titled **"CARDIAC COMPLICATIONS IN ACUTE ISCHEMIC/ THROMBOTIC STROKE PATIENTS WITH SPECIAL REFERENCE TO VENTRICULAR DYSFUNCTION"** is done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 during March 2014 to August 2014 under the guidance and supervision of **Prof. K.S.CHENTHIL, M.D.,** to be submitted to The Tamilnadu Dr. M.G.R Medical University towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in GENERAL MEDICINE.**

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CONTENTS

ABBREVIATIONS

CVA	Cerebrovascular accident
SAH	Sub arachnoid hemorrhage
ECG	Electrocardiogram
NIHSS	National Institute of Health Stroke Scale
CT	Computed tomography
MRI	Magnetic Resonance Imaging
PCD	Programmed cell death
TIA	Transient ischemic attack
ACA	Anterior cerebral artery
MCA	Middle cerebral artery
PCA	Posterior cerebral artery
LDL	Low density lipoprotein
BMI	Body mass index
AF	Atrial fibrillation

ICH	Intracranial hemorrhage
PT	Prothrombin time
EF	Ejection fraction
LVDD	Left ventricular Diastolic dysfunction
PLAX	Parasternal long axis
PSAX	Parasternal short axis
CAD	Coronary artery disease
CKD	Chronic kidney disease
HL	Hyperlipidemia
HTN	Hypertension
DM	Diabetes mellitus
HF	Heart failure

ABSTRACT

Introduction: To characterize cardiac complications occurring in acute ischemic/thrombotic stroke patients admitted to the medical emergency at a tertiary care hospital in Chennai, South India. Many extensive studies have been made in the past regarding cardiac complications in hemorrhagic stroke especially SAH.

Methods: Observational study (prospective cum retrospective) done in acute ischemic stroke patients admitted within 24 hours of symptom onset. Patients with hemorrhage in CT, age less than 18 and TIA were excluded. Electrocardiogram and Echocardiography were performed at admission and at the end of 48 hours.

Results: Hundred patients were included in this study. In our study group, 39 patients had an ejection fraction less than 50%, twenty patients had ischemic changes in the ECG, eleven patients presented with atrial fibrillation and one developed a ventricular tachycardia. Subgroup analysis revealed a higher NIHSS score among those with systolic dysfunction with ejection fraction less than 40% (10% versus 2%; $p<0.001$), atrial fibrillation on ECG (9% versus 3%; $p<0.05$), ischemic changes on ECG (17% versus 3%; $p<0.05$) compared with those without these changes.

Conclusion: A subset of acute ischemic stroke patients may have cardiac complications. Systolic dysfunction, atrial fibrillation and ischemic changes on ECG may be associated with higher in-hospital mortality rate as indirectly evidenced by the significant correlation of cardiac complications with severity of stroke. These findings support the importance of the adjunctive role of cardiac monitoring strategies in acute ischemic stroke.

KEY WORDS: Acute ischemic stroke, cardiac complications, systolic dysfunction, atrial fibrillation, Electrocardiogram.

INTRODUCTION

Any acute insult to the central nervous system has been known to cause a wide array of manifestations in the cardiovascular system. This can include asymptomatic ST-T changes, fatal or non-fatal arrhythmias, ventricular dysfunction, or cardiac dysautonomias.[1-3]

Increase in the levels of serum catecholamines following a stroke[4-6], has been thought to play a role but the intricate mechanisms involved is still an enigma. The intrinsic auto regulation of blood flow is impaired in the ischemic penumbra, making the cerebral perfusion mainly dependent on cardiac function.[7-9]Hence, cardiac dysfunction can lead to detrimental effects in acute stroke patients.

This phenomenon has been well studied in SAH by earlier investigators. But whether similar effects cause significant damage in ischemic/thrombotic stroke has been less studied.[10] Inpatients of acute ischemic stroke undergo echocardiograms to look for a cardio embolic source, but there are no recommendations pertaining to management of systolic or diastolic dysfunction in acute ischemic/thrombotic stroke.

Early identification of these cardiac manifestations might prove valuable in defining a role in the management strategies like cardiac augmentation in acute ischemic/thrombotic stroke patients.

It had been always the heart to which attention was paid in cases of stroke, either as a source of embolism or cause of hypoperfusion. Recent turn is in the emphasis towards the mechanisms involving the brain injury as a cause for cardiac dysfunctions.[11]The insular cortex had been studied and found to cause cardiac sympathetic neural upregulation and ECG abnormalities.[12-15]

Thus cardiac mortality is increased and can become a major cause of death in acute strokes.

This study is designed to augment the existing studies on cardiac dysfunction in acute ischemic/thrombotic stroke. It has been hypothesized that a considerable proportion of moderate to severe acute ischemic/thrombotic stroke have systolic and diastolic ventricular dysfunction. Alternative manifestations like arrhythmias and ST-T changes were also included. This study intends to correlate ventricular dysfunction(systolic and diastolic) with severity of acute ischemic/thrombotic stroke based on NIHSS.

AIMS AND OBJECTIVES

i) To assess the prevalence of cardiac complications in acute ischemic / thrombotic stroke patients with special reference to ventricular dysfunction.

ii) To correlate the cardiac manifestations with the severity of stroke based on NIH stroke scale.

REVIEW OF LITERATURE

CEREBROVASCULAR ACCIDENT:

Stroke is one the commonest causes of death and an important cause of disability worldwide. It is described as a syndrome of rapid onset neurological deficit which is usually focal but may be transient or permanent, the cause being vascular. It is the leading cause of neurologic disabilityin adults. The underlying vascular mechanism is ischemic in 85% of the cases. Primary hemorrhages [subarachnoid and intraparenchymal] constitute the remaining 15%.

The term transient ischemic attack (TIA) denotes an ischemic deficit that resolves rapidly (<24 hours) irrespective of the occurrence of a new infarct. Most TIAs last between 5 and 15 minutes. Recently all brain infarctions are classified as strokes regardless of duration of symptoms.

HISTORICAL PERSPESTIVE

Galen (131- 201 AD), based on dissections of animals, was the first person to described the structure of brain, its anatomy and its blood supply.

Thomas Wills, a neuro-anatomist proposed the cerebral blood vessel anastomoses at the base of brain (CEREBRAL ANASTOME), and named after him – CIRCLE OF WILLIS. He also described transient ischemic attacks, existence of occlusion of carotid artery, and the embolus as etiology for the stroke.

In 1960, Hounsfield from Britain, originated the concept of Computed Tomography (CT). In the mid-1980, MRI proved superior to CT in picking up the old hemosiderin containing hemorrhages, vascular malformation, lesion involving posterior cranial fossa, lesion abutting on bony surfaces.

At the end of 20^{th} century, advanced imaging with CT, MRI spectroscopy helped in the localizations, severity and potential reversibility of brain ischemia. These advances will help in the better management of the stroke in upcoming future.

INDIAN EPIDEMIOLOGY:

1) Incidence of stroke - 119 to 145 per lakhs of population,

2) Prevalence of stroke – 84 to 262 per lakhs of population (rural),

334 to 424 per lakhs of population (urban)

3) Cases fatality rates – around 25% (urban population), 37% (rural population) are died due to stroke and its complication during 28 days of stroke. Mortality is highest in Kolkata studies (mortality rate – 42%).

4) IV thrombolysis of stroke – 11% of the stroke

5) Stroke subtypes – 68% cerebral infarct, 32% cerebral hemorrhage

6) Stress hyperglycemia prevalence – 14 – 60%

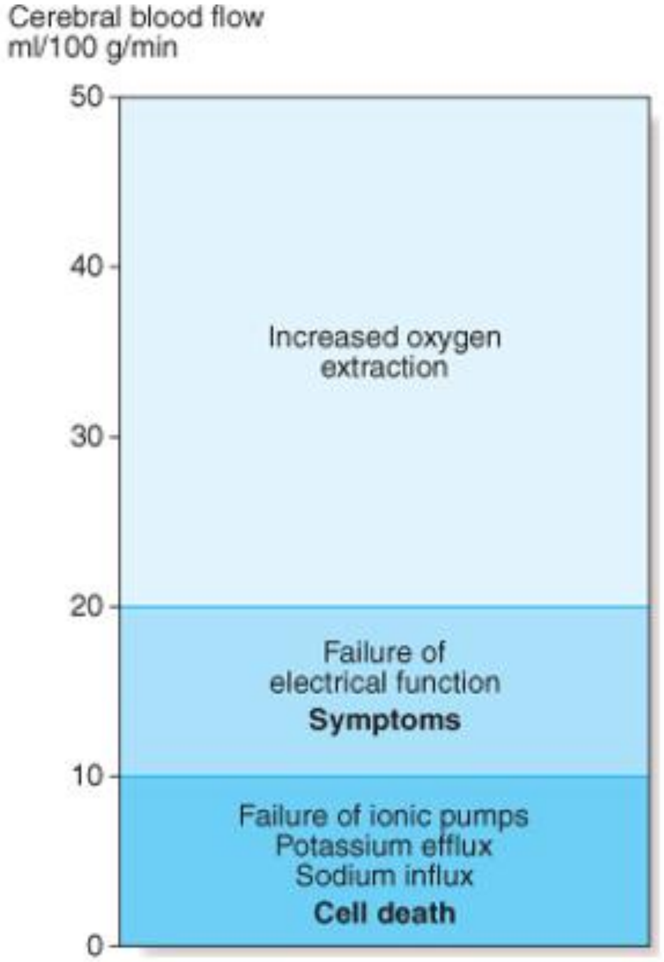
7) Stroke contributes for 1.2% of all death in the country

PATHOPHYSIOLOGY OF ISCHEMIC STROKE:

Ischemic stroke mostly results from embolic occlusion of large cerebralvessels; the major source of emboli is usually the heart or the carotid arteries. Intrinsic small-vessel disease results in small, deep ischemic lesions (lacunar strokes). Severe proximal stenosis and inadequate collaterals challenged by systemic hypotensive episodes result in low-flow strokes. The patient may experience only transient symptoms, i.e., a TIA, if blood flow is restored prior to significant cell death.

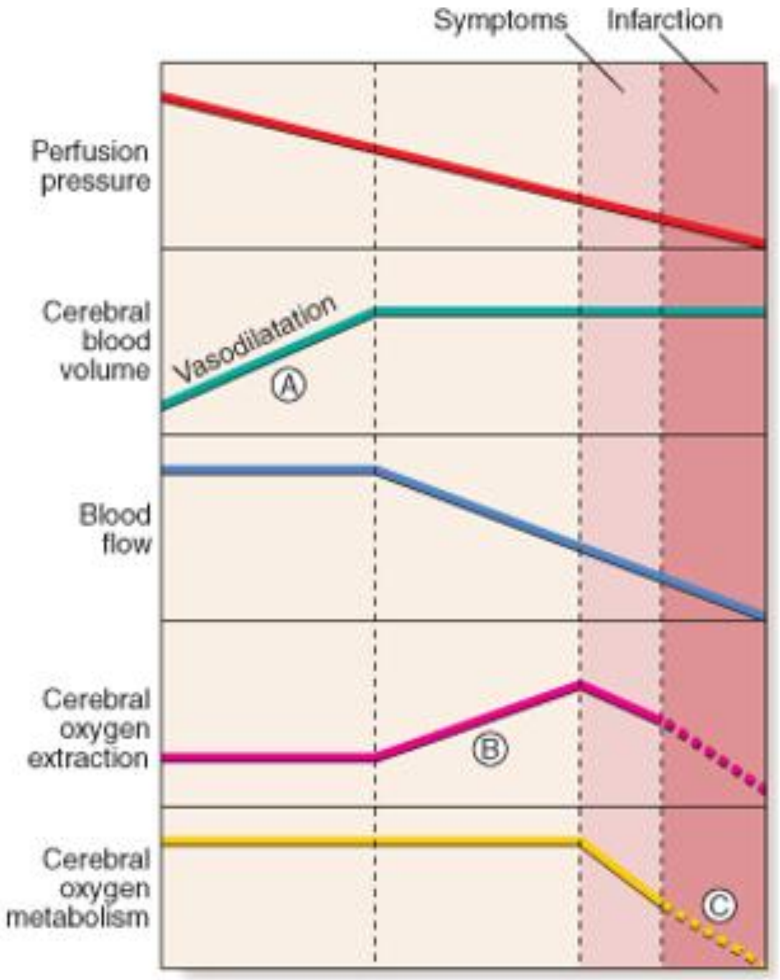
When impairment occurs in the blood flow to the brain, the neurons and other brain cells have decreased energy substrates. They ultimately die unless blood flow is promptly restored. The severity of ischemia determines the pattern of cell death. With mild ischemia, as occurring in cardiac arrest with reperfusion, certain neuronal populations are selectively vulnerable, resulting in their preferential loss. More severe ischemia causes selective neuronal necrosis, where all neurons die but glia and endothelial cells are preserved. Complete, permanent ischemia causes pannecrosis, which affects all cell types, resulting in the chronic cavitary brain lesions seen after clinical stroke.

DAMAGE SPECTRUM IN CEREBRAL ISCHEMIA



The above picture emphasizes that cerebral symptoms start to appear when the blood flow gets reduced to less than 50% causing neuronal electrical dysfunction. If this state sustains, ischemic injury to brain follows.

HOMEOSTATIC RESPONSE IN BRAIN AFTER STROKE

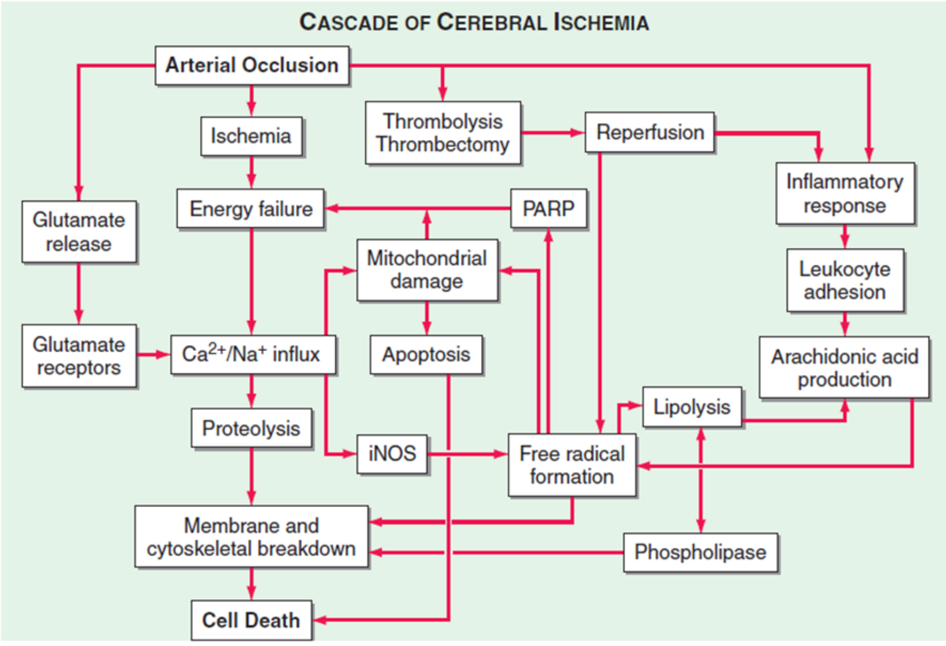


A- Vasodilation

B- Decrease in blood flow after maximal vasodilation, but increased extraction of O_2 maintains the metabolic needs

C- Further reduction in flow leading to infarction.

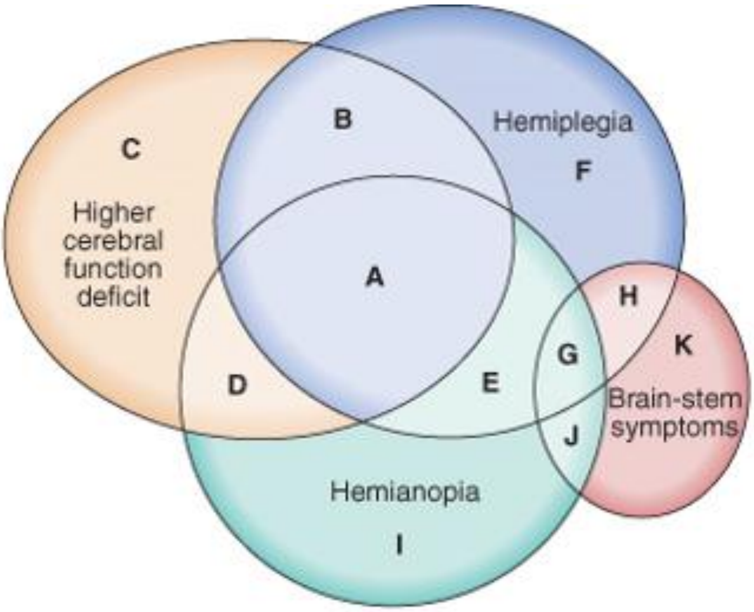
Ischemic neuronal injury is an active biochemical process which evolves over time. Lack of glucose and oxygen results in depletion of the cellular energy stores required to maintain membrane potentials and transmembrane ion gradients. Potassium leaks out of cells, leading to depolarization-induced calcium entry and also enhancing the release of glutamate through glial glutamate transporters. Synaptic glutamate causes activation of excitatory amino acid receptors, which are coupled to calcium and sodium preferring ion channels. As a result sodium enters into postsynaptic neuronal cell bodies and dendrites, causing depolarization and acute swelling. Acidosis contributes to intracellular calcium overload by activating acid-sensing ion channels. Calcium influx which exceeds the ability of the cell to buffer, sequester, or extrude calcium activates calcium-dependent enzymes (lipases, proteases, and nucleases). These enzymes and their metabolic products, such as reactive oxygen and nitrogen radicals and eicosanoids, cause the breakdown of cytoskeletal elements and plasma membranes, leading to cell death.



When ischemia is incomplete, it permits more prolonged cell survival, as in the border zone or penumbra surrounding the core of an ischemic brain region; other biochemical processes that regulate cell death are set into motion. These involve the expression of proteins engaged in programmed cell death (PCD), such as caspases (cysteine protease pro-enzyme which cleaves aspartate residues) andBcl-2 family proteins. These proteins act to cause apoptosis, a form of PCD that is distinct from necrosis and is marked by relative preservation of cell membrane integrity, cleavage of DNA into fragments of defined length (nucleosomes), margination of nuclear chromatin, blebbing of the cell membrane to form apoptotic bodies, and phagocytosis without inflammation.

Non-apoptotic forms of PCD also exist which play a role in delayed ischemic cell death.

STROKE SYNDROMES BASED ON ARTERIAL TERRITORY



A- Anterior circulation

B, C, D, E- Partial anterior circulation

F- Pure motor/lacunar

G, H, I, K – Posterior circulation

Transient ischemic attack (TIA), is defined as temporary neurological deficit caused by cerebrovascular disease, characterized by no clinical or imaging trace, with complete recovery occurring within 24 hours. Onset of stroke differs in ischemic and embolic stroke. The thrombotic stroke evolves over hours to days, in a saltatory fashion. In the embolic stroke, the onset is sudden, peak at once. The static onset & evolves over minutes is a feature in hemorrhagic stroke.

Stroke can be divided into two main subtypes – ischemic or hemorrhagic stroke. Ischemic stroke arises as a result of occlusion of blood vessels to the brain which leads to sudden cut off of the blood supply leading cerebral infarction.

Ischemic stroke is further divided into thrombotic stroke & embolic stroke. Based on underlying etiology, ischemic stroke, can arises from (1) atherosclerosis of large cerebral blood vessels, (2) occlusion of small cerebral vessel within brain parenchyma, (3) cerebral embolism. Several other causes of brain parenchyma infarction includes arterial dissection, vasculitis, hyper-coagulable state, cortical vein thrombosis.

The distinction between thrombosis and embolism is often difficult or impossible to make on clinical grounds.

Thrombotic strokes are frequently preceded by TIAs, which tend to produce similar symptoms as they affect the same territory recurrently.

Embolic strokes typically produce neurologic deficits which are maximal at onset. When TIAs precede embolic strokes, chiefly those arising from a cardiac source, symptoms characteristically vary between attacks as different vascular territories are affected.

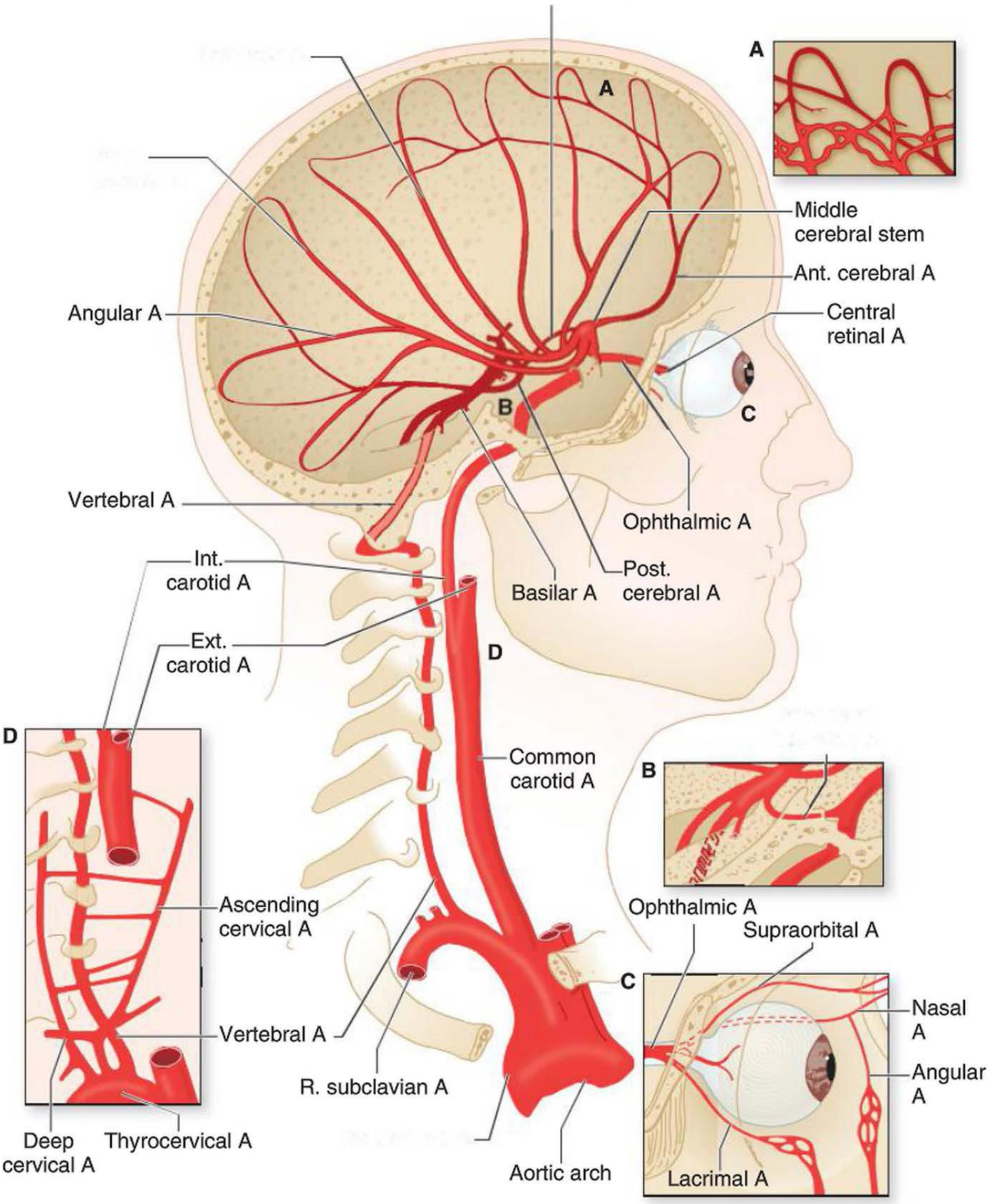
Various conditions like Migraine, Todd paralysis (seizure), Brain tumor, and abscess will be imitating stroke and these condition should be considered in differential diagnosis.

BLOOD SUPPLY TO BRAIN:

1. Internal carotid artery (Right & Left)
2. Vertebral artery (Right & Left)

Circle of Willis: is formed anteriorly by two ACA, which is connected by anterior communicating artery anteriorly, posteriorly by two PCA, communicating with anterior circulation by the posterior communicating artery.

COLLATERAL BLOOD SUPPLY IN THE BRAIN:



In the normal situation, the anterior two-third of the cerebral circulation is supplied by the internal carotid artery and the posterior one – third is supplied by the vertebral artery. In the cases of blood vessels occlusion, collateral develop distal to the site of occlusion, and the collateral development depends on the vessels occluded, and whether the other artery are free of disease or not.

Other regions of collateral blood flow are:

1. Leptomeningeal anastomoses
2. Around the orbit
3. Parenchymal anastomes

Venous drainage of the brain:

1. Superficial cerebral veins

2. Deep cerebral veins

Both these veins drains into the dural venous sinuses, which further drains into the internal jugular vein. The cerebral veins lacks valves, they are thin walled, & the blood flow in these veins are in the same direction as that of the neighboring arteries

RISK FACTORS

Risk factors can be fixed or it can be modified through preventive measures. Based on that there are two groups of risk factors.

1. NON -MODIFIABLE:

a) Age: the most important risk factor of the stroke, &the risk doubles after 55 years, for every decade.

b) Sex: incidence and mortality of the stroke is higher in male gender compared to that of female gender.

c) Genetic factors: chromosome 12 polymorphism

d) Race/Ethnicity: Afro-Caribbeans >Asians >Europeans have stroke incidence in the decreasing order

2. MODIFIABLE:

a) Diabetes mellitus: 1.8 to 3.5 times increases in the stroke incidence, with most of them are lacunar strokes. Hyperglycemia and insulin resistance are the other risk factors.

b) Systemic hypertension: the most important risk factor in the development of ischemic stroke. The incidence is three times higher when compared to normotensive people. Recent studies revealed that 46% reduction in the stroke incidence for every 7.5 mmHg reduction in diastolic BP. Around 40% of strokes have a blood pressure of greater than 140mmHg.

c) Dyslipidemia (elevated LDL cholesterol, elevated triglycerides): Statin therapy reduce the stroke incidence by 20 -30%, because of its action of stabilizing the plaque, anti-thrombotic action, improved endothelial function and by reducing the inflammation.

d) Cigarette smoking: increases the risk of all types of stroke. The risk is highest in the younger group, with risk proportionate to the number of cigarettes smoked.

e) Alcohol intake: the risk is variable. Risk is reduced with low to moderate consumption. Higher level of consumption increases the incidence of hemorrhagic stroke.

f) Obesity: along with smoking, a BMI of > 25 kg/m2, account for 60% of stroke, in the group of men up to sixty five years

g) Transient ischemic attack (TIA): 10 times higher risk of stroke when compared to person without stroke.

h) History of Stroke in the past.

i) Asymptomatic carotid bruit/stenosis

j) Cardiac illness: the presence of left ventricular hypertrophy by ecg increases ischemic stroke risk by 10 fold, presentation of congestive heart failure increases stroke incidence by nine fold. Atrial fibrillation raises the risk of embolic stroke by 5 to 7 times than age matched population of normal cardiac rhythm. Other risk factors include mitral valve prolapsed, prosthetic valves, endocarditis, and congenital heart diseases.

k) Aortic arch atheromatous diseases

l) Increased fibrinogen level

m) Raised homocysteine level

n) Decreased serum folate

o) Increased anti-cardiolipin antibodies

p) Oral contraceptive use: risk is higher in young women who is taking estrogen content of more than 50 mcg. OCP increases ischemic stroke secondary to enhance platelet aggregability & alteration in clotting factors.

q) Anticoagulation therapy: increases risk of hemorrhagic stroke.

r) Intake of low potassium, reduced serum potassium level.

CLINICAL FEATURES:

General physical examination may reveal obesity, feeble or absent peripheral arterial pulsations, vascular bruits, unequal or raised blood pressure, postural hypotension and retinopathy.

Prodromal warning symptoms of TIA may precede in about 60% of the patients. Such episodes of TIA are often brief(lasting for a few minutes to less than an hour) and may come singly or in successive spells over a number of hours or days or months, and leave no significant residual signs.

TIAs may not be always related to posture or the level of BP, and may disappear altogether. However, in some cases (10% to

15%),an evolving or a full-blown stroke may follow the last ischemic spell.

When the stroke evolves in a stepwise manner, (‘thrombosis in evolution’) the symptoms may appear in each limb in succession or simultaneously. This stuttering or intermittent progression is typical of atherothrombosis. Not infrequently, the stroke may announce itself abruptly as a single major catastrophic event(accomplished infarction or completed stroke). The other clinical manifestations are based on where the artery is blocked.

The focal signs and symptoms accompanying ICH reflect the location of the hemorrhage and are indistinguishable from ischemia occurring in the same vascular territory. Lobar hemorrhages frequently produce contralateral weakness or sensory loss, language disturbance, hemianopia or lesser field disorders, and parietal lobe signs. Their relationship to the cortex makes them more likely to be complicated by seizures. Seizure are more frequent in ICH, occurring in as many as a quarter of individuals, although they are also seen in about 5% of patients with ischemia, particularly early after onset.

Occasionally, very small hematomas present with symptoms indistinguishable from transient ischemic attacks (TIAs).However, majority of ICHs cause complete stroke.

Putaminal hemorrhages typically cause contralateral hemiparesis with variable degrees of sensory loss, ataxia and with larger hematomas, a homonymous hemianopia.

Thalamic hemorrhages can result in contralateral sensory loss and weakness, while if they extend to or compress the superior midbrain, they may result in depressed signs. Pontine hemorrhages often result in reduced consciousness, pinpoint pupils, bilateral weakness and pontine cranial nerve dysfunction, and may be severely disabling.

Cerebellar hemorrhages are particularly important to identify clinically as they may require surgical intervention, which can be life-saving. The onset can be deceptive, with initial nonspecific brainstem symptoms (e.g. vertigo or double vision),followed a few hours or even days later by progressive clinical features, including gait, trunk or limb ataxia, nystagmus, headache, vomiting and coma from brainstem compression. Hemiparesis will be rare. Posterior circulation stroke has an unpredictable clinical course at the time of onset. An abrupt deterioration may occur after an initial stable course. Moderate degrees of quadrigeminal cisternal compression predicts poor outcome unless the hematoma is evacuated early in the course. Severe mass effect on the cistern carries a poor prognosis.

WATER SHED INFARCTS:

It occurs in the border region between two arterial territory. These infarct are common during or after the surgery, severe arterial hypotension after cardiac arrest, prolonged hypoxemia, and bilateral severe carotid artery diseases.

Ischemia in the border zone of the ACA, MCA & PCA manifest in the form of bilateral parieto-occipital infarcts with clinical features of visual disturbances, optic ataxia, cortical blindness and difficulty in predicting the size, distance and movement.

Unilateral severe arterial occlusion or stenosis will leads to unilateral watershed infarct when these is some degree of hemodynamic failure in these patients. This can also occur in the situation of microembolism or the hyperviscosity states.

Ischemia between ACA & MCA region results in bilateral upper limb cortical sensorimotor impairment (man-in-barrel), impaired saccadic eye movements due to involvement of frontal eye fields.

MCA & PCA territory involvement results in bilateral parieto temporal infarction with cortical blindness, defect in the verbal & the nonverbal material dyslexia, dyscalculia, dysgraphia.

Watershed infarct can also occur in the regions of PICA, AICA & SCA., and also involving internal watershed region in the centrum semi ovale near to & slightly above the body of lateral ventricles.

INVESTIGATIONS:

Routine studies include CXR and ECG, blood tests, and imaging. Further studies of coagulation are indicated if a hypercoagulable state is suspected.

IMAGING IN STROKE

1. **Non Contrast CT**:

This is the diagnostic brain imaging study in the initial stroke evaluation.

(A) Hyperacute infarct (<12 hours): in this stage, there will be early changes in the form of grey-white matter differentiation will be lost, sulcus & Sylvian fissure effacement &obscuration of the lentiform nucleus. Dense MCA sign- Horizontal part of the middle cerebral artery may appear hyper dense, even before the appearance of infarct.

(B) Subacute infarct (24-48 hours):

Wedge shaped area of decreased attenuation involving both the grey and with matter in a typical vascular territory. The initial mass effect then begin to decrease in 7-10 days.

(C) Chronic infarct:

Well delineated, focal areas of encephalomalacia appears in CT scans.

Adjacent sulci become prominent and the ventricles on the ipsilateral side enlarges. Enhancement disappears after eight to ten weeks.

Scan negative infarct: early scans may be negative in 60% of cases within 12 hours of ictus, after 2 -3rd week after infarct (blooming).

2. Multimodal Ct:

i) Whole brain perfusion CT- identify the cerebral blood volume and areas of hypottentuation representing the ischemic core.

ii) Dynamic perfusion CT – provides absolute measures of cerebral blood flow, mean transit time and cerebral blood volume.

iii) Helical angiography – noninvasive method to assess the vasculature of both the intra-cranially and extra- cranially and identify the stenosis or occlusion of the vessels.

3**. Magnetic Resonance Imaging (MRI):**

i) Superior in detecting ischemia, when compared with CT.

ii) In acute infarct, DWI is highly sensitive in the first few hours after infarction. The diffusion restriction in acute infarct will appears as high intensity in DWI images with low ADC signal and appears dark, and during the initial stages T2 weighted images are normal.

4. Multimodal MRI:

i) Diffusion weighted MRI (DWI) – identify ischemic regions within minutes of symptom onset and early identification of lesion size, site & age.

ii) Perfusion weighted MRI (PWI) – provides relative measures of cerebral hemodynamics. The ischemic penumbra is approximated on MRI as region of perfusion change without a corresponding diffusion abnormality (diffusion perfusion mismatch)

5. MRI angiography:

Delineates blood flow & vascular lesions, including atheromatus plaques in the carotid and vertebrabasiliar systems. Identify the larger blood vessel abnormality better than that of the distal lesions.

6. Other brain imaging:

Includes oxygen-15 Positron emission tomography (PET); Xenon enhanced CT brain; Single photon emission computed tomography (SPECT) identifies thresholds of reversible ischemia; transcranial Doppler sonography for evaluation of the blood flow velocity and patency of the main intracranial arteries.

7. **Duplex Doppler ultrasonography:**

Detection of the carotid artery atheromatous plaques and obstruction of the carotid artery.

8. Echocardiography to assess the potential causes of TIA or evolving stroke.

BLOOD TESTS IN PATIENTS WITH ISCHEMIC STROKE / TIA:

i) Hemoglobin
ii) Hematocrit
iii) WBC count (and differential if abnormally high or low)
iv) Total platelets
v) aPTT
vi) PT-INR
vii) Serum fibrinogen level

viii) Blood sugar

ix) Serum calcium

x) Total cholesterol, HDL, LDL.

xi) Blood urea nitrogen

xii) Electrolytes (sodium, chloride, potassium, and carbon dioxide)

xiii) Homocysteine

xiv) C-reactive protein

xv) Erythrocyte sedimentation rate (ESR)

TREATMENT:

ACUTE ISCHEMIC STROKE

Treatments designed to reverse or lessen tissue infarction in acute ischemic stroke include:

(1) Medical support,

(2) Thrombolysis and endovascular techniques,

(3) Antiplatelet agents,

(4) Anticoagulation and

(5) Neuroprotection.

MEDICAL SUPPORT:

It optimizes perfusion in ischemic penumbra surrounding the infarct.

- Blood pressure should never be lowered precipitously (exacerbates the underlying ischemia), and only in the most extreme situations should gradual lowering be undertaken (e.g., malignant hypertension with BP> 220/120 or, if thrombolysis planned, BP> 185/110 mmHg).
- Maintenance of Intravascular volume with isotonic fluids. Osmotic therapy with mannitol may be necessary to control edema in large infarcts, but isotonic volume must be replaced to avoid hypovolemia.
- In cerebellar infarction (or hemorrhage), rapid deterioration can occur from brainstem compression and hydrocephalus, requiring neurosurgical intervention.

THROMBOLYSIS AND ENDOVASCULAR TECHNIQUES:

- IV rTPA is used for acute ischemic strokes of less than three hours duration without any evidence of hemorrhage. Recent data support the use of IV rtPA for deficits of 3-4.5 h duration.[55]

ANTIPLATELET AGENTS:

- Aspirin in doses upto 300mg has been found safe and is beneficial in acute ischemic stroke.

ACUTE INTRACEREBRAL HEMORRHAGE:

- Rapidly identify and correct any coagulopathy.
- Stuporous or comatose patients generally are treated presumptively for elevated ICP. Treatment for edema and mass effect with osmotic agents may be necessary; glucocorticoids not helpful.
- Neurosurgical consultation should be sought for possible urgent evacuation of cerebellar hematoma; in other locations, data do not support surgical intervention.

COMPLICATIONS AFTER STROKE:

ACUTE COMPLICATIONS

- Raised intracranial pressure and herniation
- Aspiration and pneumonia

COMPLICATIONS OF IMMOBILITY

- Pneumonia
- Contractures
- Deep vein thrombosis
- Bed sores
- Urinary tract infections
- Constipation

LATE COMPLICATIONS

- Depression
- Epilepsy
- Thalamic pain
- Social problems

NATIONAL INSTITUTESOF HEALTH STROKE SCALE (NIHSS)

It is a scale used to quantify objectively the severity of a stroke. NIHSS has 11 parameters. Each parameter has a score between 0 and 4 with 0 denoting normal function and higher scores indicating higher level of impairment. Scores of each parameter are added up to derive at the patient's NIHSS score. Range of NIHSS is 0(minimum) to 42(maximum).

The scale is a simple bed side tool which is reliable and can be easily used by doctors and paramedics. Many studies have shown that increased NIHSS score was associated with an increased risk of neurological and medical complications.

The NIHSS is an excellent and validated tool to predict patient outcomes. NIHSS scores correlate well with the volume of damaged brain.

The components of the NIHSS is as the following:

ITEM	SCORE
1. a) Level of consciousness	
- Alert	0 points
- Drowsy	1 point
- Stupor	2 points
- Coma	3 points
b) Response to 2 questions (orientation)	
- Know age and current month	0 points
- Answers 1 question correctly	1 point
- Cannot answer either question correctly	2 points
c) Response to 2 commands	
- Follows 2 commands correctly	0 points
- Follows 1 command	1 point
- Cannot follow either command	2 points

2. Best gaze (movement of eyes to left or right)

- Normal eye movements	0 points
- Partial gaze paresis to one side	1 point
- Forced gaze palsy to one side	2 points

3. Visual fields

- No visual loss	0 points
- Partial homonymous hemianopia	1 point
- Complete homonymous hemianopia	2 points
- Bilateral visual loss	3 points

4. Facial motor function

- No facial weakness	0 points
- Minor unilateral facial weakness	1 point
- Partial unilateral facial weakness	2 points

- Complete paralysis of 1 or both sides 3 points

5. (a and b) Upper-extremity motor function (left and right scored independently 0-8 points)

- Normal movement 0 points
- Drift of upper extremity 1 point
- Some effort against gravity 2 points
- No effort against gravity but moves 3 points
- No movement 4 points

6. (a and b) Lower-extremity motor function (left and right scored independently 0 – 8 points)

- Normal movement 0 points
- Drift of lower extremity 1 point
- Some effort against gravity 2 points
- No effort against gravity but moves 3 points
- No movement 4 points

7. Limb ataxia (cannot be tested in presence of paresis)

- No limb ataxia	0 points
- Ataxia present in 1 limb	1 point
- Ataxia present in 2 limbs	2 points

8. Sensory function

- No sensory loss	0 points
- Mild-to-moderate sensory loss	1 point
- Severe-to-total sensory loss	2 points

9. Language

- Normal language	0 points
- Mild-to-moderate aphasia	1 point
- Severe aphasia	2 points
- Mute	3 points

10. Articulation

- Normal articulation	0 points

- Mild-to-moderate dysarthria 1 point
- Severe dysarthria 2 points

11. Extinction or inattention (neglect)

- No neglect or extinction 0 points
- Visual or sensory inattention or extinction 1 point
- Profound inattention to visual and sensation 2 points

Score	Stroke Severity
0	No Stroke Symptoms
1-4	Minor Stroke
5-15	Moderate Stroke
16-20	Moderate to Severe Stroke
21-42	Severe Stroke

NEUROCARDIOLOGY

It had been always the heart to which attention was paid in cases of stroke, either as a source of embolism or cause of hypoperfusion. Recent turn is in the emphasis towards the mechanisms involving the brain injury as a cause for cardiac dysfunctions.

When an acute ischemic/thrombotic stroke happens in any patient with underlying heart disease, the damage is severe. The auto regulation of blood flow is lost in the ischemic penumbra as the main factor which determines it, ‘the cardiac function’ is under stake.

Over the past fifty years lot of studies had been done to demonstrate various cardiac complications occurring in acute stroke. These include ECG changes like QT_c prolongation, ST-T changes, septal U waves, LAD. These changes were more observed after a hemorrhagic stroke especially SAH, [16-22] but also seen to occur in ischemic strokes.

A number of neurosurgical studies have shown that ECG abnormalities and left ventricular dysfunction (wall motion hypokinesias) can occur in hemorrhagic stroke especially SAH.[23-26] Also described are myocardial stunning and myocardial necrosis. Other findings were increased levels of natriuretic factors, catecholamines in the plasma. Myocardial perfusion too gets affected regionally.[27-30]

The insular cortex had been studied and found to cause cardiac sympathetic neural upregulation and ECG abnormalities.[31]In a recent animal study it has been found that the left insular cortex related to parasympathetic cardiac and vasomotor function whereas the right insula is concerned with the sympathetic cardiac and vasomotor control.

Also, SAH on the right side involving the Sylvian fissure had been found to be associated with cardiac complications.[32-36]Thus any insult to the right insula either directly or indirectly can lead to consequences like sudden cardiac death after stroke.[56-59]

Regardless of the presence of pre-existing heart disease, ST segment and T wave changes, QT prolongation, and ventricular and supraventricular tachyarrhythmias are common ECG manifestations of stroke. In addition, greater variability of systolic blood pressure has been noted in patients with acute stroke compared with controls.

Centrally mediated sympathetic hyperactivity, reduced cardiac parasympathetic innervation and abnormal baroreceptor function have been proposed to be the causes for these findings. In animal studies it has been shown that the levels of circulating catecholamines are elevated during acute stroke.[37-39]

Reactive oxygen species are abundantly generated during acute ischemic stroke. There is a good body of evidence indicating that oxidative stress is a pivotal mediator of tissue injury in acute ischemic stroke.

Cerebral autoregulation is the phenomenon by which maintaining the cerebral blood flow at a relatively constant rate despite variations in perfusion pressure. The mechanism by which auto-regulation occurs is not well understood, and may involve multiple pathways.

There are evidences which suggests that the smooth muscle in cerebral vessels can respond directly to changes in perfusion pressure, contracting when pressure increases and relaxing when pressure decreases. Reductions in cerebral blood flow may lead to cerebral vasodilatation due to release vasoactive substances, although the substance responsible for this have not been identified. Nitric oxide which releasing from vascular endothelium appears to play a role in autoregulation.

Abnormal Calcium Modulation in Heart Disease:

Heart failure and cardiac hypertrophy can lead to systolic and diastolic dysfunction. One of the important mechanisms is defects in intracellular calcium modulation.

There is a highly positive correlation between end-diastolic calcium levels and diastolic relaxation abnormalities. The duration of diastole is prolonged in most models of congestive heart failure. This change has been found to be associated with transient intracellular calcium. However, marked increase in systolic and diastolic calcium occurs in myocardial ischemia and yet this is not coupled with the chronotropic effects which gets decreased. Hence, ischemia and decreased responsiveness of myofilament to calcium may affect both systolic force generation and diastolic relaxation.

Sarcolemmal receptors and mechanisms:

The cardiac sarcolemma is a complex structure that contain multiple receptors (adrenergic and others), channels exchanges and pumps that are necessary for maintaining cellular homeostasis.

Aspects of normal sarcolemmal function are impaired in the failing heart.

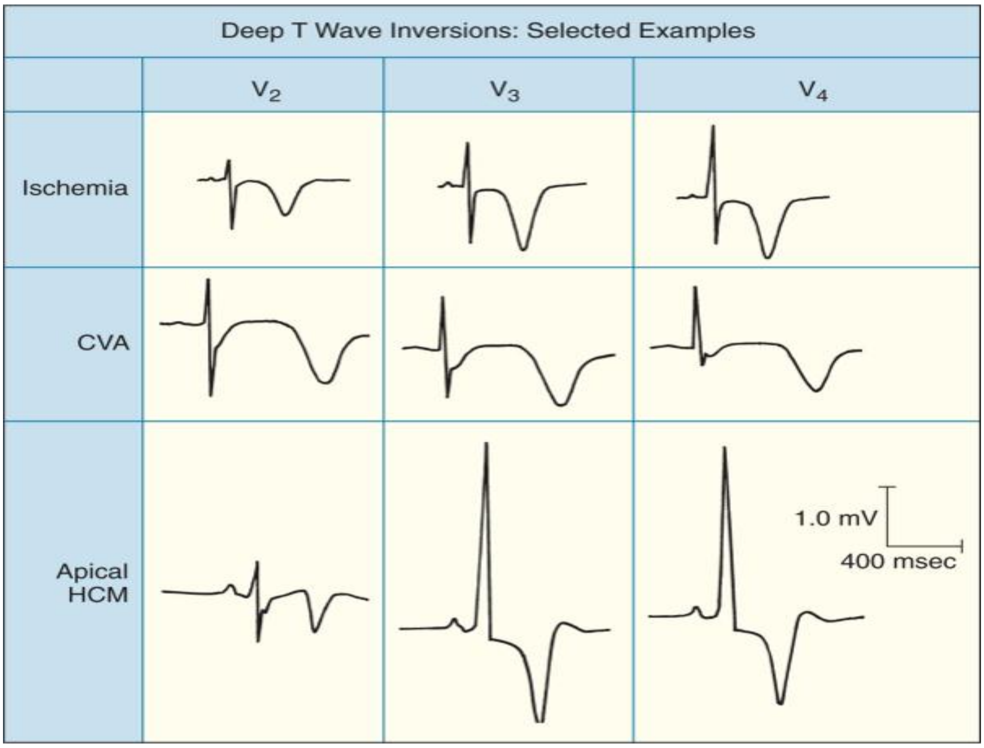
Synergistic interactions between an increase in Cytosolic Calcium ($Ca(I)^{2+}$) and a decrease in adenosine triphosphate (ATP) availability increase diastolic tension and can cause an exacerbate diastolic dysfunction.

Effect of hormones:

Weber et al had presented an interesting study incriminating aldosterone as one of the stimulus of myocardial fibrosis. Brain natriuretic peptide levels have been found in relation to the decrease in LV mass index as well as diastolic function. In Hypertensive patients, this peptide can be an indicator of ventricular remodeling. The octapeptide angiotensin II definitely plays a central role in LV remodeling by promoting the growth of cardiac myocyte.

Renin angiotensin system and endothelial system may play a role in the development of hypertrophy, ventricular fibrosis and eventually diastolic failure. This had been found to occur in hypertensive patients. Endothelin system also has a role in causing this effect. Thus, there is robust evidence suggesting that acute brain injury alters the autonomic regulation of the cardiovascular system.

T-WAVE CHANGES DUE TO VARIOUS CAUSES



- The deep T wave inversions are to be differentiated among various causes.
- The T wave inversion in the picture row of CVA is caused by an SAH and is characterized by the marked prolongation of QT interval.

ATRIAL FIBRILLATION

Recent data suggest that a focal mechanism plays a role and it involves both increased automaticity and multiple re-entrant wavelets which occurs mainly in the left atrium around the pulmonary veins.

Characteristically, P waves are absent. There is chaotic atrial activity and fibrillatory (F) waves are present.

Ventricular rhythm is usually irregularly irregular. If AF is suspected with a regular ventricular response then the possibility of a heart block with junctional or ventricular escape should be considered.

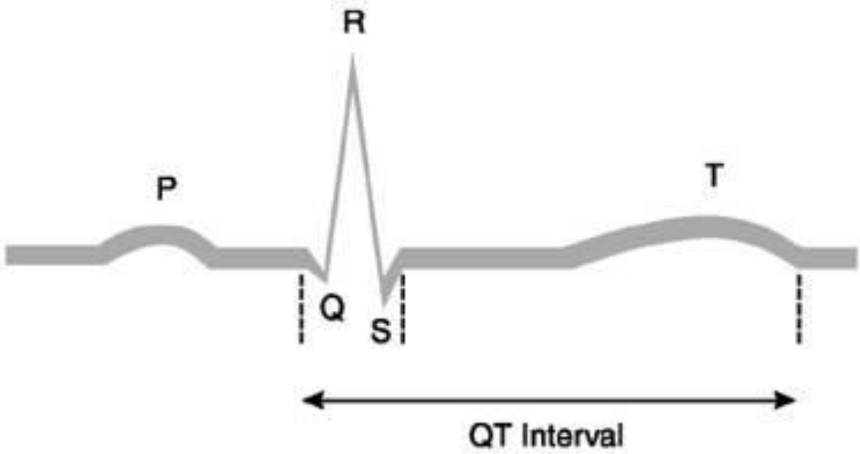
Common causes of AF include

- Old age
- Hypertension

- Valvular heart disease
- Congestive heart failure
- Coronary artery disease
- Hyperthyroidism
- Drugs
- Chronic lung disease
- Post cardiac surgery

QT INTERVAL

In a standard surface electrocardiogram the QT interval includes the QRS complex, ST segment and the T wave corresponding to phases 0 to 3 of action potential.



QT dispersion has been defined as inter lead variability (difference between maximum and minimum QT interval). Increased QT dispersion is a direct reflection of disparity in myocardial recovery. Thus determination of QT dispersion may help to predict arrhythmogenicity in patients with acute cerebrovascular accident.

Homogeneity of ventricular recovery is considered to be protective against ventricular arrhythmias. On the contrary, dispersion of recovery is considered to be arrhythmogenic.

Measurement of QT interval and QT dispersion:

QT interval is measured from beginning of QRS complex to the end of T wave. The presence of a U wave is not included in measuring QT interval.

Corrected QT interval:

The QT interval is affected by heart rate. It is longer when the heart rate is slow and shorter when the heart rate is fast, so the QT interval should be corrected for heart rate.

The most frequently used formula for correcting QT interval for heart rate is the Bazett's formula. Corrected QT interval is QT interval (in seconds) divided by the square root of preceding RR interval (in seconds).

A prolonged QT interval is defined as

>0.44 seconds in men

>0.46 seconds in women and children

>0.50 seconds (if there is bundle branch block or intraventricular conduction delay).

Various studies have found that QT dispersion varies between 30 and 60 milliseconds in normal population. A QTcd of more than 70 milliseconds is considered abnormal.

ECHOCARDIOGRAM

INTRODUCTION:

Echocardiogram is safe, non-invasive, and repeatable and allows visualization of structures and thus assesses the anatomical and hemodynamic activity in the cardiovascular system.

Echo has become the most important tool for diagnosis of structural and functional abnormalities of the heart. Anatomic details can be accurately portrayed and cardiac structures can be measured and their movements traced throughout the cardiac cycle.

COMPONENTS

- TRANSDUCER: is a probe housing piezoelectric elements. These elements have the property of changing shape when electrical current is applied and vice versa. Phased array sector transducer is used.
- TRANSMITTER: produces the impulse that is to be sent to the transducer. Also known as 'pulser'.
- RECEIVER: receives the current produced in the transducer from the sound energy that returns.

- AMPLIFIER: amplifies the signals which returns and processes them for display in the monitor.

ULTRASOUND WAVE:

- Ultrasound travels through soft tissues of the body at particular speeds and as it travels ultrasonic wave is progressively attenuated.
- The ultrasound wave is either reflected, scattered or absorbed depending on the properties the different tissues in the body.
- Frequency is number of cycles in one sec (i.e) number of times the piezoelectric crystal vibrates (expands and contracts).
- Period is the length of time to complete one cycle.
- Relationship between transducer frequency and tissue penetration

  -Penetration is inversely related to frequency.

RESOLUTION:

As in the field of optics resolution is defined as the smallest distance between two points at which they can still be depicted as separate.

Any area of interest may be displayed both in longitudinal and cross-sections.

The quantity of the image produced depends on the image resolution.

TYPES

-Axial resolution**:** of the transducer is spatial resolution along the axis of the sound beam, also known as range, depth or longitudinal resolution.

-Lateral Resolution**:** is transverse resolution and is perpendicular to the axis of the sound beam, also known as azimuthal and angular resolution and depends on beam diameter.

MODES OF DISPLAY:

The echo returning to the transducer may be presented on the display in several different forms.

- A-mode: Amplitude mode echoes are presented as vertical spikes on a baseline the height of the spike represents the amplitude of the returning echoes and bases line represents the depth.

- B-mode: Brightness mode. The brightness of the dot represents the amplitude of the returning echoes.

- M-mode: is the motion mode. The two display parameters in an M-mode trace are distance from transducer and time. The pattern of movement represents the motion of the structure over a specific time interval.

- 2-D Mode: 2 dimensional display of scanned anatomy providing tomographic views. But reduction in temporal resolution, requires M-mode to complement this mode in cases of fast moving structures and precise timing of events.

- Doppler principle is used for assessment of blood flow.

DOPPLER ECHOCARDIOGRAPHY

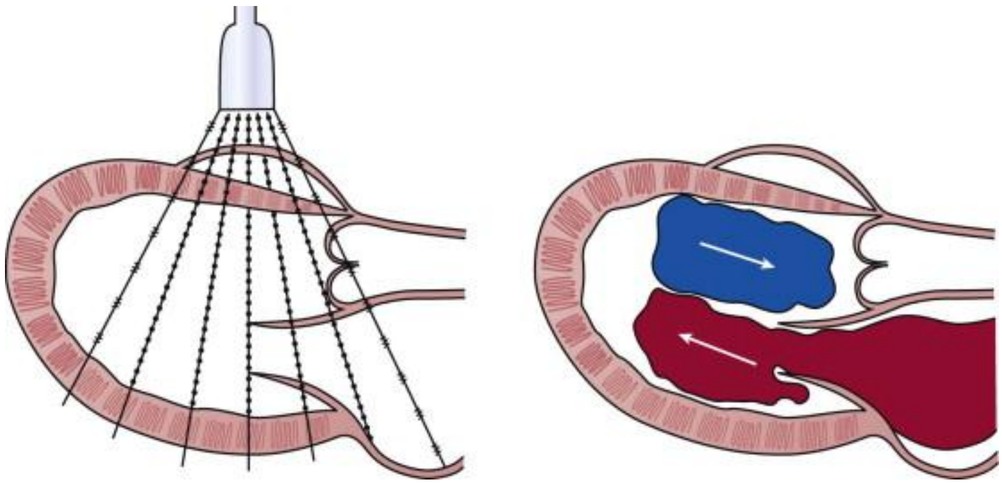
A modality that gives a hemodynamic assessment of the heart.

Based on Doppler principle that the frequency of sound increases or decreases as it moves closer or farther from the observer.

- PW (pulsed-wave) and CW (continuous wave) are spectral analysis modes. PW mode is used to assess low velocity flow in specific areas. CW mode is used to assess high velocity flow
- Color flow imaging. Helps assess intracardiac blood velocity.

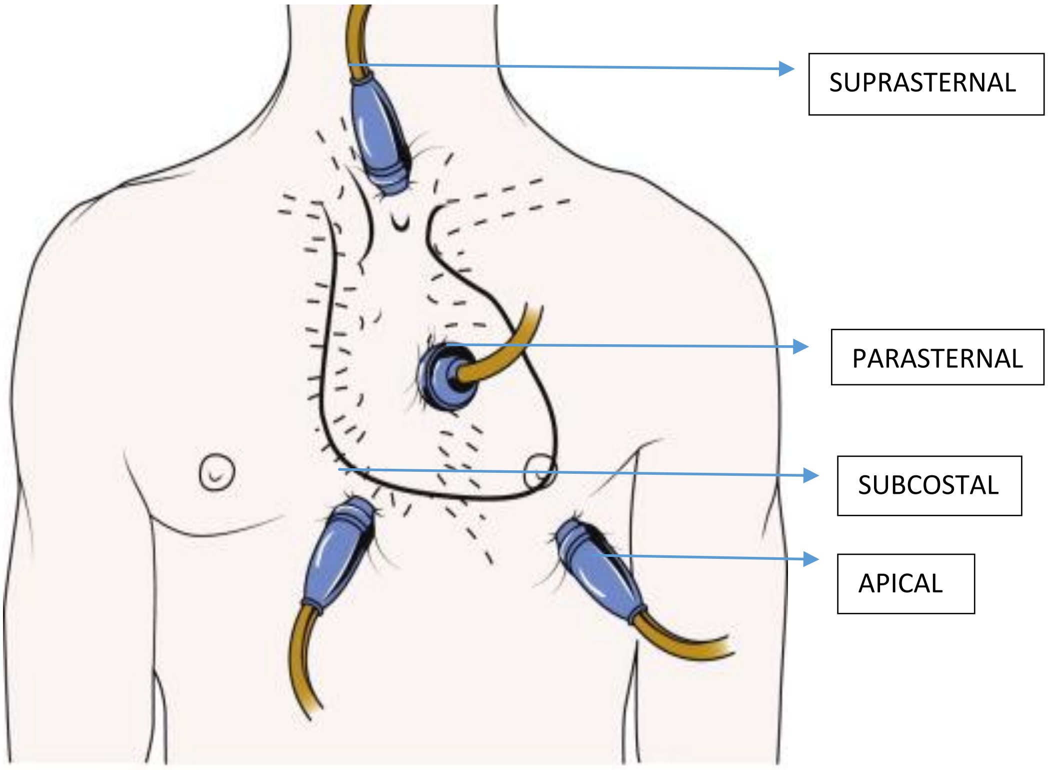
- TDI (Tissue Doppler Imaging) Used as an adjunct in assessing LV diastolic function.
- CMM (colour M mode)

COLOURDOPPLER



The dots indicate multiple sampling sites (gates). The frequency shift measured at each gate is correlated automatically (autocorrelation) and converted toa preset color scheme (red for flow toward and blue for flow away from the transducer).

DIFFERENT VIEWS USED IN ECHOCARIOGRAPHY



PARASTERNAL VIEWS

Left sternal border approach is called parasternal view. Long and short axis views are taken in the parasternal position.

Long Axis (PLAX):

Transducer beam positioned to slice the heart in its long axis from the apex to its base. Marker is directed towards the right shoulder of the patient. This view demonstrates the long section through the

LV, LA, mitral leaflets, aortic root, aortic cusps and a small section of the RV.

Short Axis (PSAX):

This place is perpendicular to the LAX plane and is obtained by rotating the transducer 90^0 from the LAX plane and the marker is directed towards the left shoulder of the patient.

APICAL VIEWS

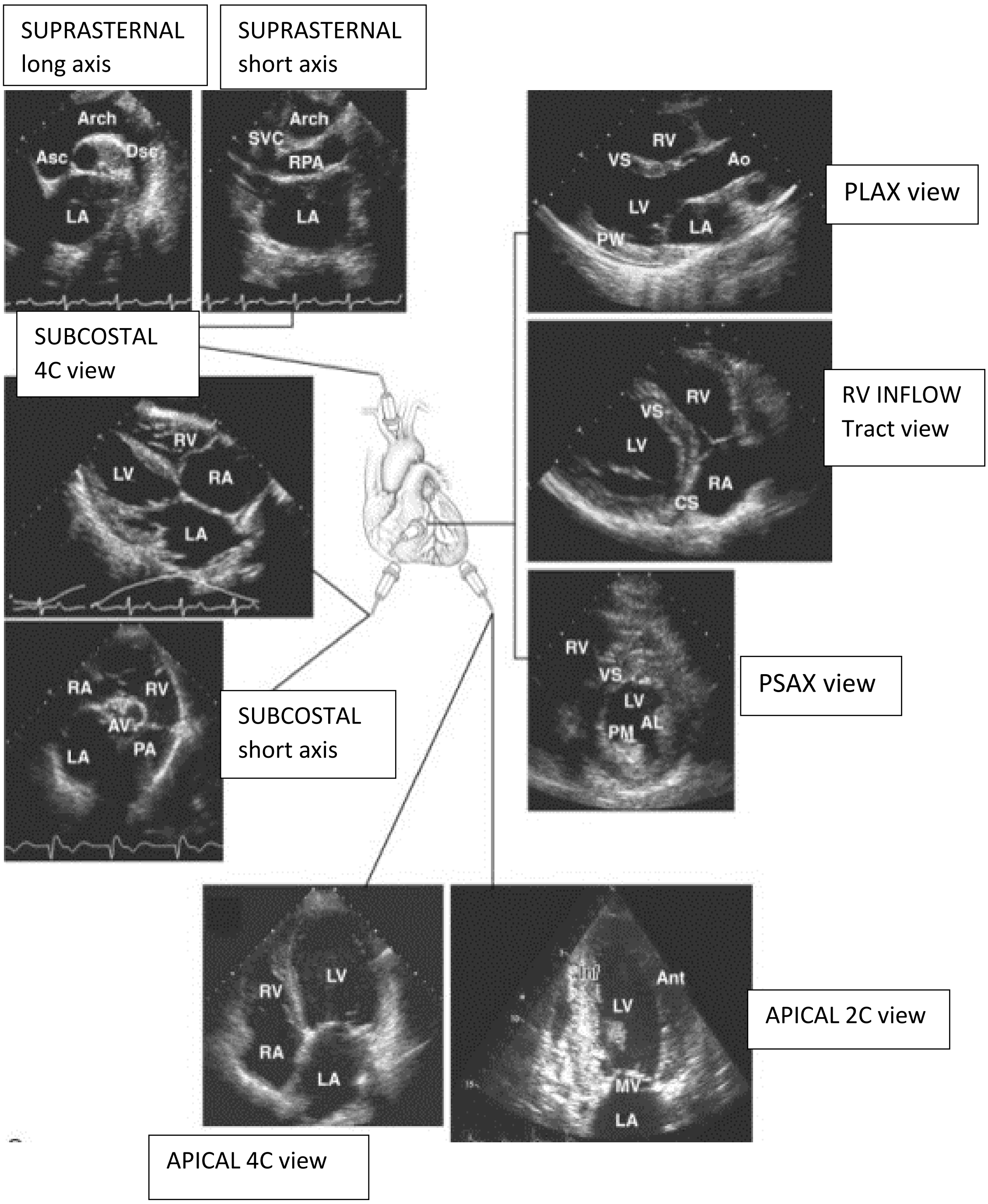
Apical 4 – Chamber View:

Obtained by placing the transducer at the apex, with the marker directed towards the left shoulder and beam parallel to the plane of the left shoulder and right hip.

Apical 5 – Chamber View:

Obtained with the transducer in the apical position and tilting the transducer antero-superiorly, thus visualizing the LV outflow tract and the aortic root.

SUMMARY OF VARIOUS VIEWS



Apical 2-Chamber View:

Obtained by rotating the transducer 90^o anti-clockwise, from the routine 4 chamber view.

SUBCOSTAL VIEWS:

It has two views namely four chamber and short axis view

SUPRASTERNAL VIEWS:

The transducer is placed in suprasternal notch to see the aortic arch and its branches. The marker is directed towards the left ear of the patient.

It again has a long and short axis viewing

M-MODE EXAMINATION:

M-mode records cardiac motion at a high frame rate, thus helps better appreciation of subtle changes in valvular or wall motion.

COLOR DOPPLER:

Makes it possible to visualize and quantify blood flow in the heart. Colors represent direction & velocity of blood flow.

EVALUATION OF SYSTOLIC AND DIASTOLIC FUNCTION

SYSTOLIC FUNCTION

There are many parameters which could be used to express systolic function by echocardiography.

- LV Ejection fraction
- Stroke volume & Cardiac index
- Fractional shortening
- Systolic tissue velocity of the mitral annulus and myocardium
- Strain
- Regional wall motion defects

LV Ejection fraction

It is the most commonly used parameter to assess to assess global LV function.

It has many limitations, yet it is very good prognostic marker in various cardiac diseases and it is useful in determining the optimal treatment strategies in a given clinical cardiac condition. It has a significant inter-observer variability. Hence should be performed more objectively as far as possible using the following volumetric parameters.

$$EF = \frac{\text{(LV end-diastolic volume - LV end-systolic volume)}}{\text{LV end-diastolic volume}}$$

Fractional shortening

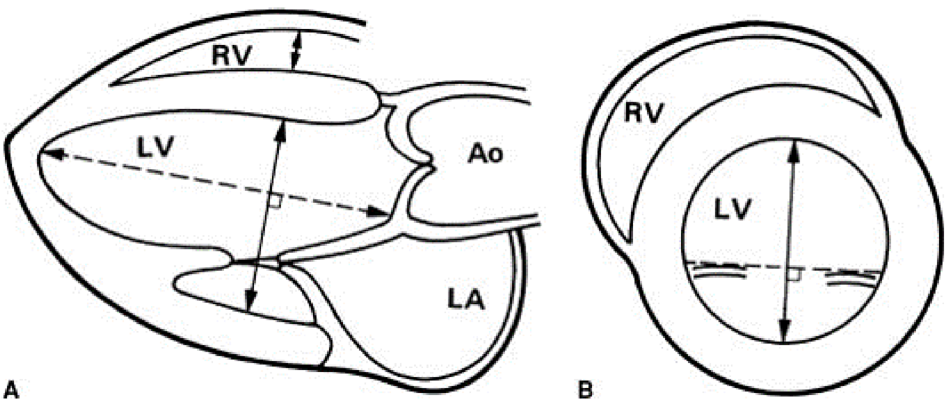
It is the change in the LV dimension percentage with each contraction of the ventricle.

$$FS = \frac{\text{(LVend-diastolic dimension – LV end-systolic dimension)}}{\text{LV end-diastolic dimension}}$$

Stroke volume

It can be measured by obtaining the difference between LVEDV and LVESV measured by Simpson's method or 3D echo.

MEASUREMENT SITES OF LEFT VENTRICULAR DIMENSION (American society of Echocardiography)

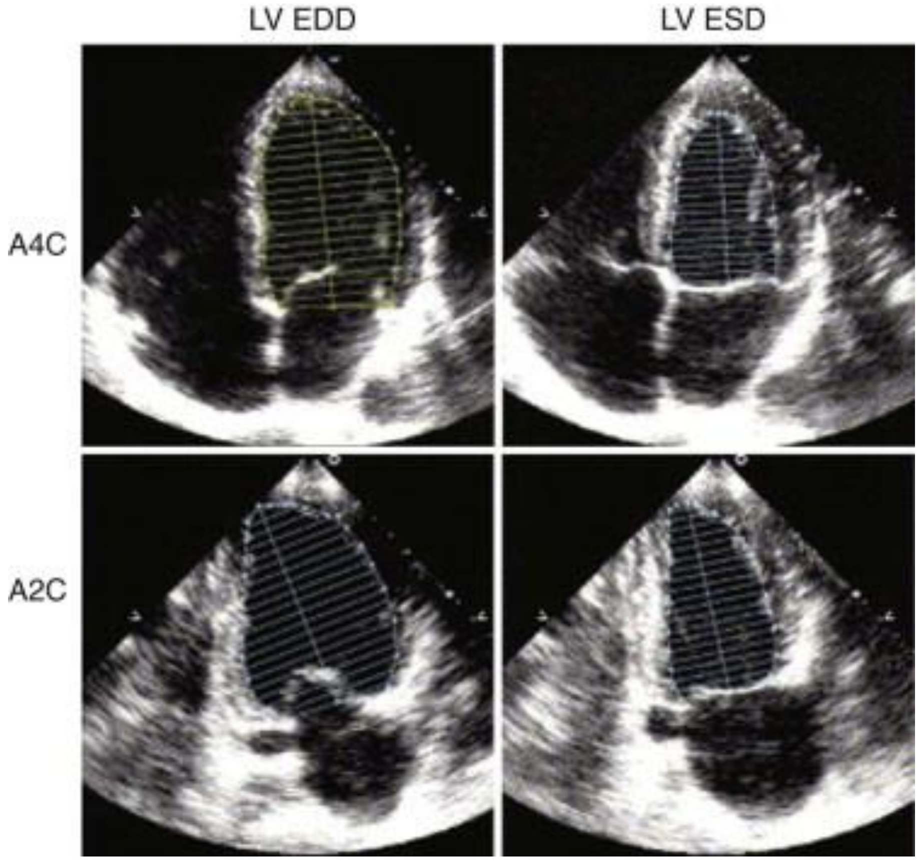


PLAX view

PSAX view

Simpson's method

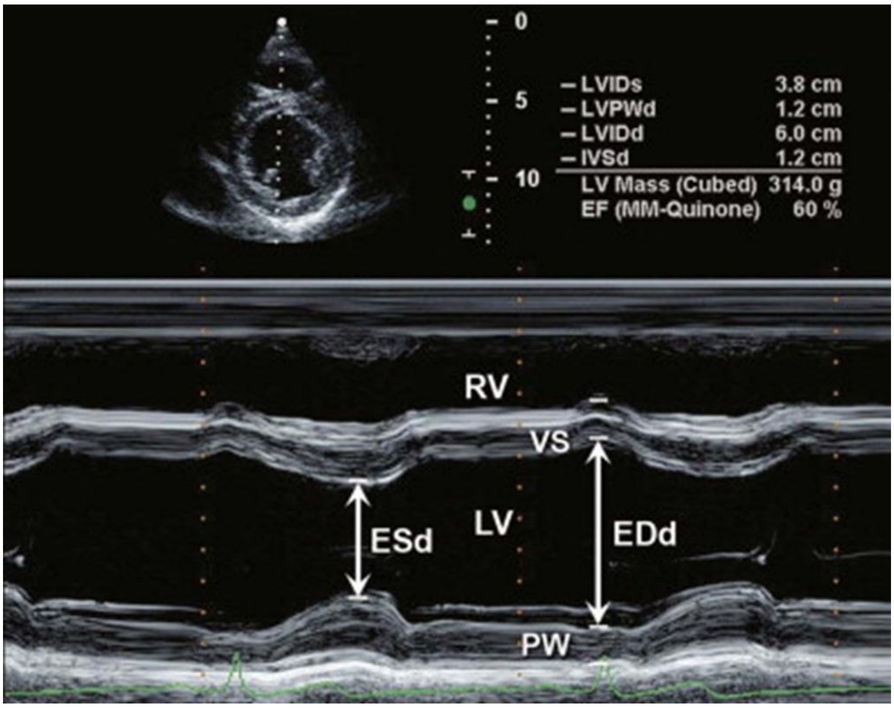
Measurement of LV volume by the biplane Simpson method needs two orthogonal apical views, namely the 4C and 5C views. The left ventricle is divided into 20 cylinders or discs. Their volumes are calculated and added to provide LV volume at end diastole (LV EDD) and end systole (LV ESD).



LVEDD- left ventricular end diastolic dimension

LVESD- left ventricular end systolic dimension

TWO-DIMENSIONAL GUIDED M-MODE ECHOCARDIOGRAM OF LEFT VENTRICLE (LV) AT THE PAPILLARY MUSCLE LEVEL



In this echo window, the LV end-diastolic internal dimension (EDd) measured at the onset of QRS is 60 mm, and the LV end-systolic internal dimension (ESd) is 38 mm.

Ef is calculated from the formula discussed above.

DIASTOLIC FUNCTION

Assessment of diastolic function is important because almost half of the patients with heart failure can have a normal LVEF.

Echocardiography using 2D, M-mode and Doppler are useful in determining the diastolic function.

STEPS TO ASSESS DIASTOLIC DYSFUNCTION

1. Evidence of LVDD in M-mode and 2D echo. Abnormal relaxation leads to decrease in the slope (M-mode), decreased motion of the mitral annulus during early diastole and increase in the size of left atrium
2. Velocity at the mitral inflow. This reflects transmitral pressure gradient and useful in staging diastolic dysfunction.
3. Myocardial relaxation (by TDI), Mitral annulus velocity (E').
4. Mitral inflow velocity (E and A). E-early diastole ; A-late diastole

GRADING OF DIASTOLIC DYSFUNCTION

Grade:1 (mild dysfunction)-Ventricular relaxation is impaired but filling pressures are normal. Hence Mitral E velocity is decreased and A velocity is increased, producing an E/A ratio of less than 1, with prolonged DT(Dispersion time).

Grade:2 (moderate dysfunction)- also called as pseudonormalized mitral flow filling pattern. Due to this the E/A is 1 to 1.5 and DT normal (160 to 240 ms). This pseudonormalisation happens because of raised LA pressure.

How to differentiate this from true normal?

1. E’<7
2. Presence of mid-diastolic flow
3. Increase in A with Valsalva manouvere

Grade: 3-4 (severe diastolic dysfunction) –also called as restrictive filling. It can be caused by any condition leading restrictive physiology.

E/A ratio >2, and shortened DT (<160 milliseconds)

Intraventricular relaxation time IVRT (<70 milliseconds)

MATERIALS AND METHODS

SETTING OF THE STUDY:

This study was conducted at the Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

ETHICS COMMITTEE APPROVAL:

Obtained from the Institutional ethics committee.

STUDY POPULATION:

Patients admitted with acute ischemic/thrombotic stroke in the medical wards of our Institute.

STUDY DURATION:

This study was conducted over six months.

SAMPLE SIZE:

Hundred consecutive patients admitted with acute ischemic/thrombotic stroke.

TYPE OF STUDY:

Observational (prospective and retrospective)

INCLUSION CRITERIA:

Patients presenting within 24 hours of symptom onset and diagnosed with acute ischemic/thrombotic stroke.

EXCLUSION CRITERIA:

1. Age younger than 18 years
2. Evidence of cerebral hemorrhage on initial head CT
3. Resolution of neurologic symptoms within 24 hours
4. Presence of documented chest pain

SPONSORSHIP:

No sponsorship was obtained to perform this study.

CONFLICTS OF INTEREST:

No

DISCLOSURES:

No disclosures to mention, related to this study.

DATA COLLECTION AND METHODS:

- Patients were selected carefully based on the inclusion and exclusion criteria
- Informed consent was obtained from each patient or the relative
- The clinical details were obtained based on a unique proforma (enclosed) prepared for this study
- Clinical history was taken based on a questionnaire in the proforma
- Patients were subjected to routine blood investigations (CBC/RFT/LFT), plain CT brain, 12 lead Electrocardiogram and Echocardiography at admission.
- 12 lead Electrocardiogram and Echocardiography were repeated at 48 hours of admission.
- During their stay, patients were monitored for arrhythmias and hemodynamic changes
- LV systolic dysfunction and LV diastolic dysfunction, and ECG changes including ST-T changes (ischemic/non-ischemic) and arrhythmias, were taken as the prime variables in the study.[60]

- During echocardiography, systolic dysfunction was assessed with Four-chamber view which was used to record the LV volume and Ejection Fraction.

- Systolic dysfunction was assessed based on Ejection fraction. Though this is could miss LV dysfunction with preserved ejection fraction, our aim in this study is only to detect early cardiac changes during acute stroke.

- Diastolic dysfunction was assessed using Doppler echocardiography by integrating information obtained from pulsed-wave Doppler of mitral inflow and pulmonary venous flow combined with 2D assessment of left atrial size.

- Patients were grouped based on NIH stroke scale.

- All the data were pooled from the proforma and master chart prepared.

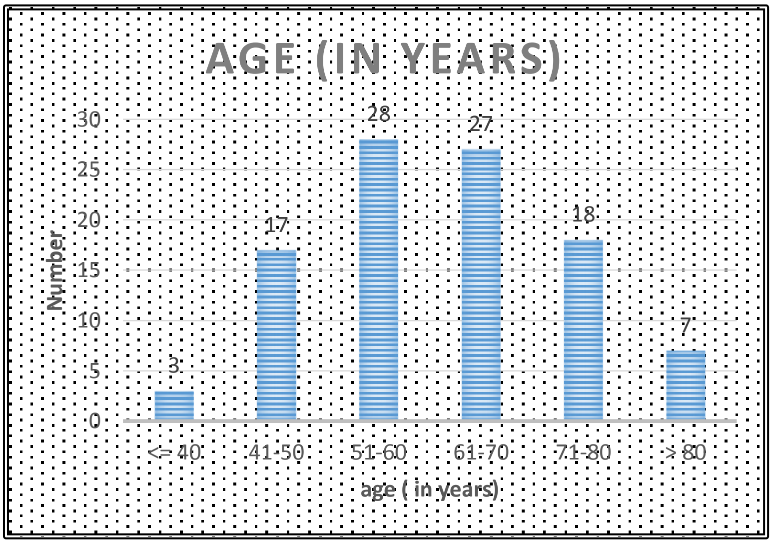
- Data were analyzed using SPSS package and chi square tests.

OSERVATION AND RESULTS

As the sample size in our study is 100, separate mention about the percentages are made only where relevant.

Table: 1 AGE DISTRIBUTION

Age group	Frequency
<= 40	3
41-50	17
51-60	28
61-70	27
71-80	18
> 80	7
Total	100



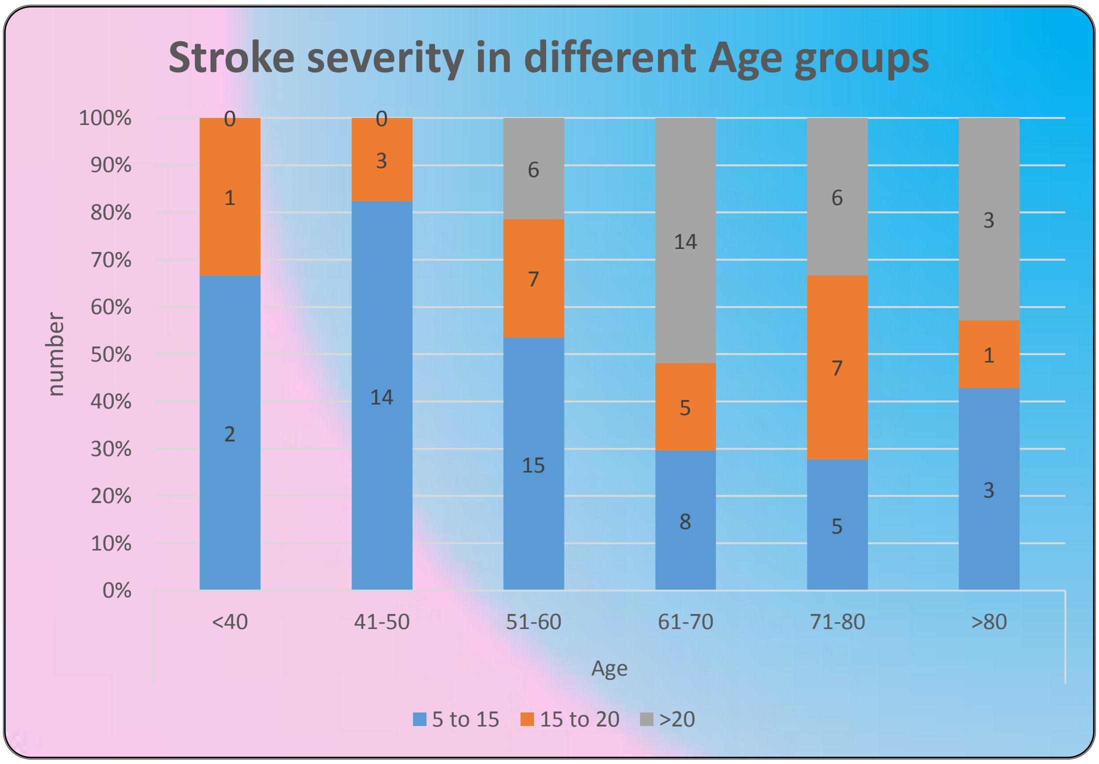
Majority of the population in the study fall in the 51 to 70 years age group.

The age distribution in our study takes the bell shaped curve pattern. To mention here again, age group less than 18 years were excluded from our study.

Table: 2 STROKE SEVERITY IN DIFFERENT AGE GROUPS

	Age (in years)						P-value
NIHSS	**<40**	**41-50**	**51-60**	**61-70**	**71-80**	**>80**	
5 to 15	2	14	15	8	5	3	0.012*
15 to 20	1	3	7	5	7	1	
>20	0	0	6	14	6	3	

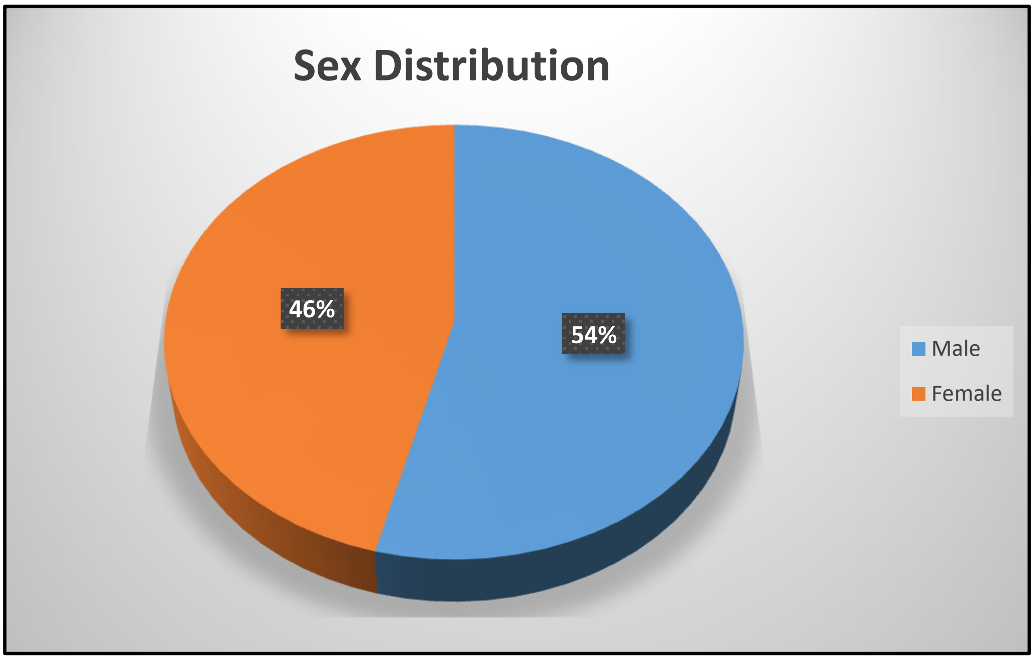
* - significant at 5 level



The 50 to 70 age group has got higher proportion of 'severe stroke' group. Again, it is this age group which forms the bulk of our study.

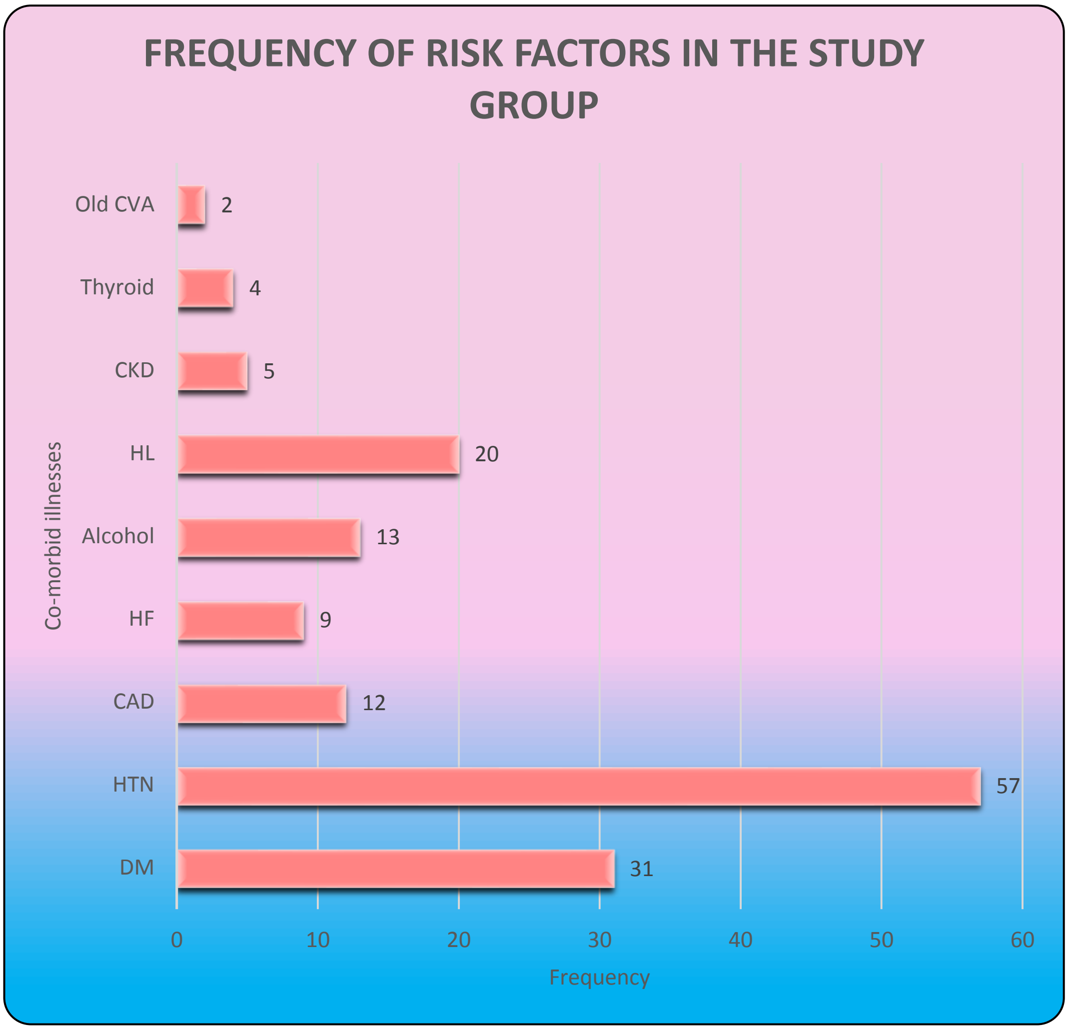
Table: 3 SEX DISTRIBUTION

SEX		NIHSS		
		5-15	**16-20**	**> 20**
Sex	**Male**	25	13	16
	Female	22	11	13
Total		47	24	29



The study population group is fairly divided with sex. There was no significant difference in the stroke severity among sexes.

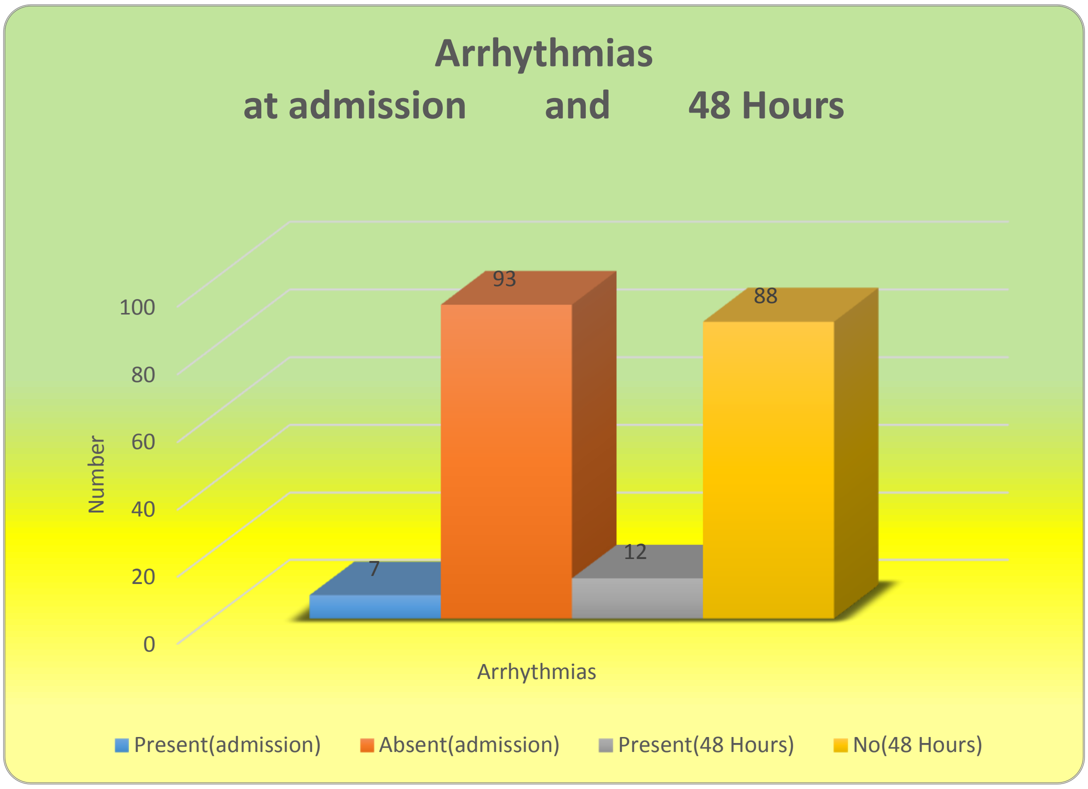
Table: 4 FREQUENCY TABLE OF RISF FACTORS



In our study group, Hypertension was seen in 57% and Diabetes mellitus was seen in 31% of the patients. Hyperlipidemia (20%) and Alcohol consumption (13%) were the other common risk factors observed.

Table: 5 ARRHYTHMIAS (AT ADMISSION AND 48 HOURS)

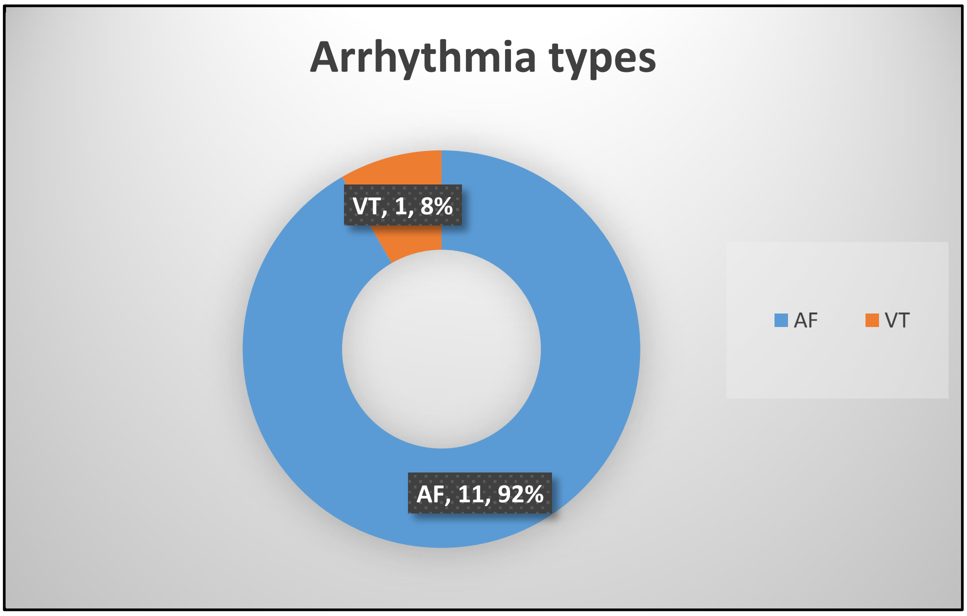
	ARRHYTHMIAS	
NUMBER	**admission**	**48 hours**
present	7	12
absent	93	88



A small proportion of 7% had arrhythmias at admission. The number increased to 12% at 48 hours of monitoring. This increase is although not statistically significant.

Table: 6 ARRYTHMIA TYPES

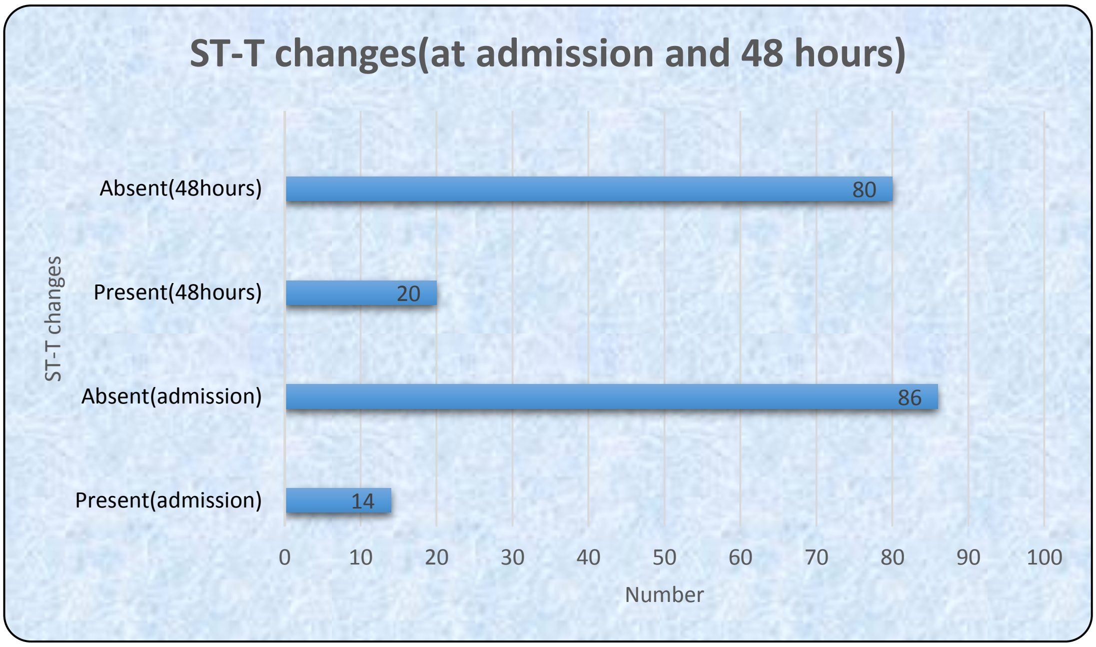
Type Of Arrhythmia	Number	Percentage
AF	11	92%
VT	1	8%



Among the 12 patients who had rhythm disturbances, 11(92%) had atrial fibrillation and 1 patient (8%) developed ventricular tachycardia.

Table: 7 ST-T CHANGES (AT ADMISSION AND 48 HOURS)

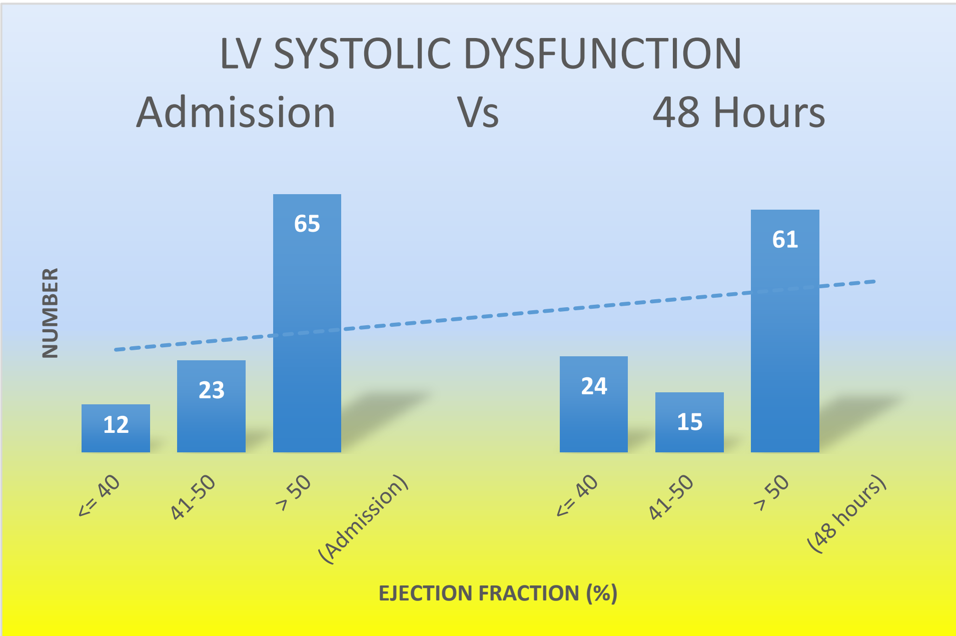
	ST-T CHANGES	
NUMBER	**admission**	**48 hours**
present	14	20
absent	86	80



In our study group, 14% had ST-T changes in the ECG at admission. This included only ischemic ST-T changes excluding non-specific ST-T changes andST depression &T wave inversions seen in acute strokes. At 48 hours, 20% had these changes in the ECG.

Table: 8 LV SYSTOLIC DYSFUNCTION (AT ADMISSION AND 48 HOURS)

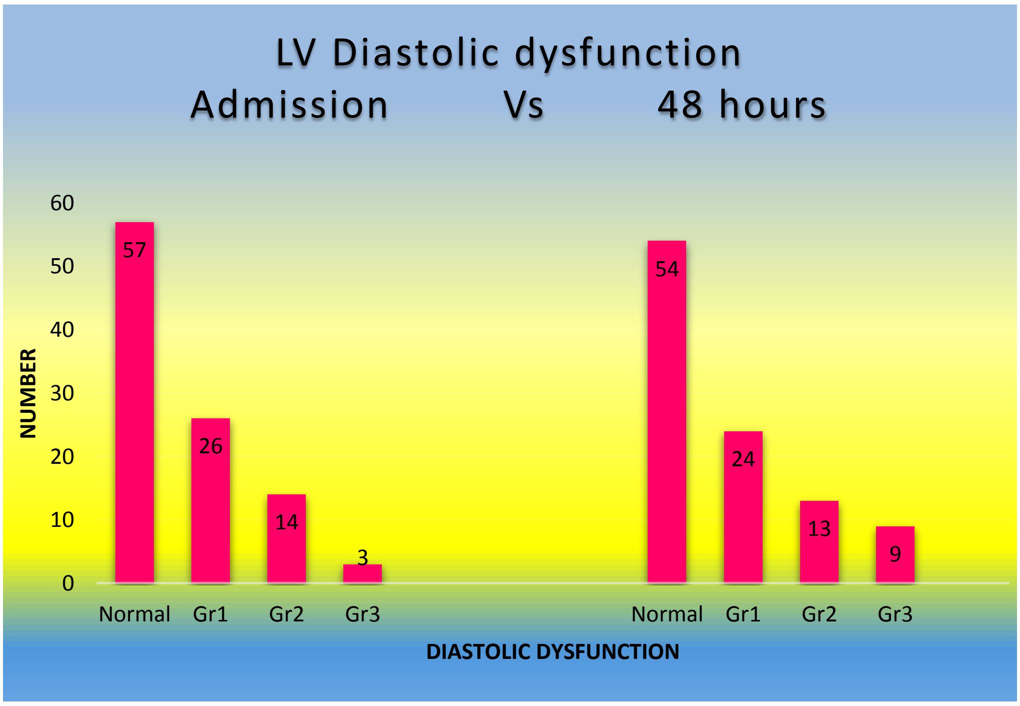
	LV systolic dysfunction	
Ejection Fraction	**admission**	**48 hours**
<40	12	24
41-50	23	15
>50	65	61



In our study, 12% had EF<40% at admission and this number doubled to 24% at 48 hours echocardiography. 23% fell in the EF 41-50% group at admission and 15% at 48 hours. More than 60% had normal LV systolic function based on the EF both at admission and 48 hours.

Table: 9 LV DIASTOLIC DYSFUNCTION (AT ADMISSION AND 48 HOURS)

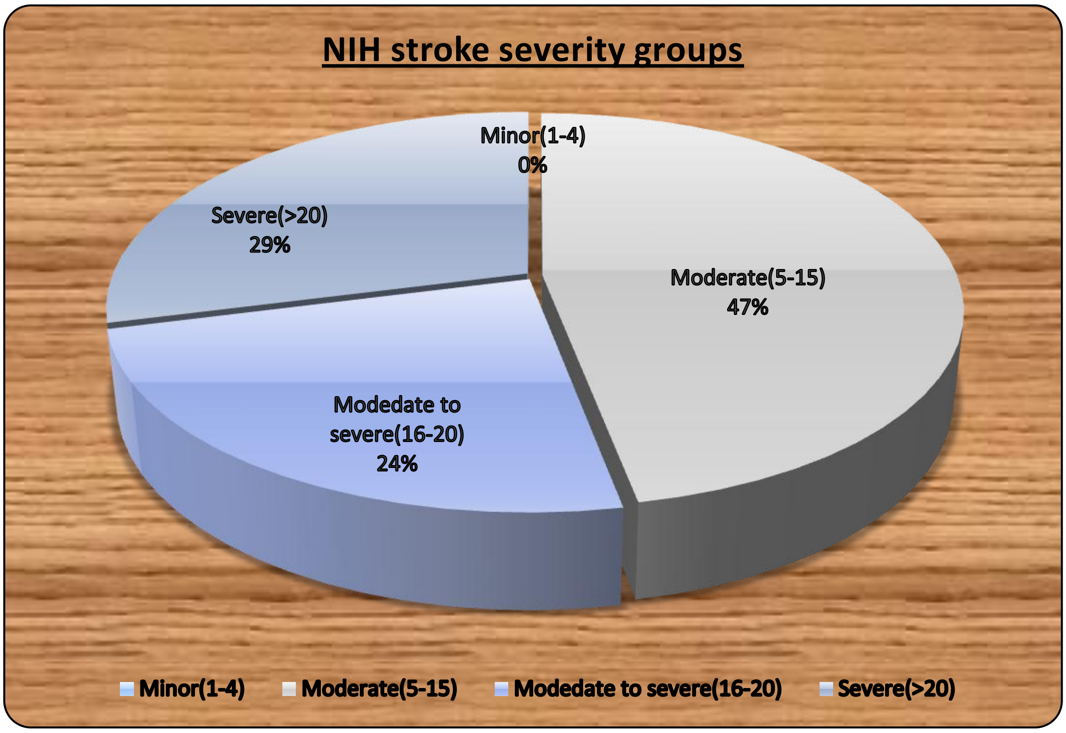
	LV diastolic dysfunction	
Grade	**admission**	**48 hours**
normal	57	54
1	26	24
2	14	13
3	3	9



In our study, 3% had Gr.3 LVDD at admission and the number rose to 9% at 48 hours. Around 25% had Gr.1 LVDD. Nearly half had normal LVDD.

Table: 10 NIH STROKE SEVERITY GROUPS

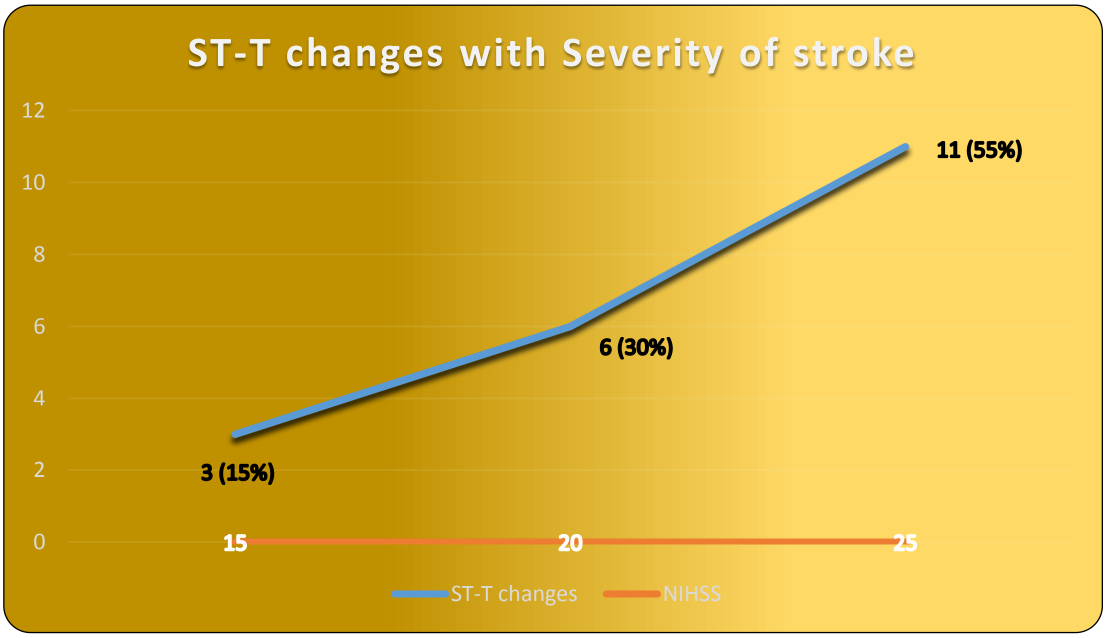
NIH stroke scale		frequency
5-15	Moderate	47
16-20	Moderate to severe	24
>20	Severe	29
	Total	100



In our study, 47% patients had a moderate stroke, 24% had a moderate to severe stroke, and 29% had a severe stroke.

Table: 11 ST-T CHANGES WITH SEVERITY OF STROKE

ST-T CHANGES		NIHSS			
		5-15	**16-20**	**>20**	**Total**
	present	3	6	11	20
	absent	44	18	18	80
					100

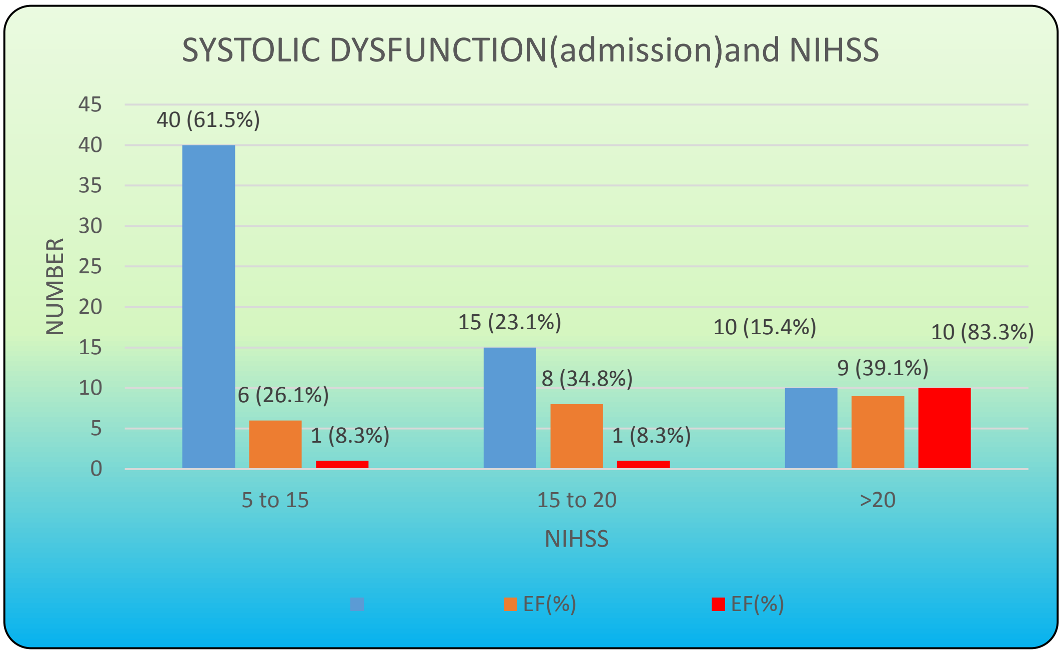


In our study, 20 % had ST-T changes at the end of 48 hours and among them more than half (55%) belonged to the severe stroke group. Six patients (30%) belonged to the moderate to severe stroke group.

Table: 12 SEVERITY OF LV SYSTOLIC DYSFUNCTION (at admission) AMONG STROKE SEVERITY GROUPS

		NIHSS				p value
Ejection Fraction (%)		**5-15**	**16-20**	**>20**	**total**	
	>50	40	15	10	**65**	
	41-50	6	8	9	**23**	
	<40	1	1	10	**12**	**<0.001****
					100	

****- highly significant at 1 level.**



In our study, a total of thirty five patients had some degree of systolic LV dysfunction during admission.

In the systolic dysfunction group, twelve patients had an EF of less than 40% and ten (83.3%) amongst them belonged to the severe stroke group which is statistically highly significant.

P value obtained was <0.001

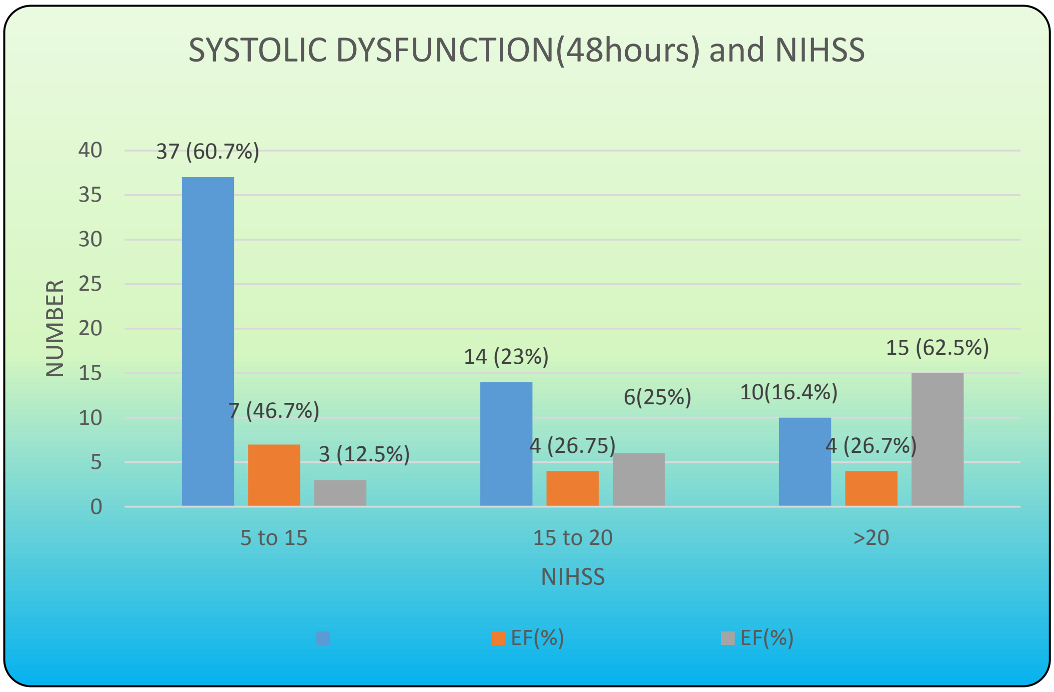
Twenty three patients had mild LV systolic dysfunction and the proportion of them in severe stroke group was more though not statistically significant.

Sixty patients had normal LV systolic function and forty (61.5%) among them belonged to the moderate stroke group and this difference among severity groups is statistically highly significant.

Table: 13 SEVERITY OF LV SYSTOLIC DYSFUNCTION (at 48 hours) AMONG STROKE SEVERITY GROUPS

Ejection Fraction (%)		NIHSS				p value
		5-15	16-20	>20	total	
	>50	37	14	10	61	
	41-50	7	4	4	15	
	<40	3	6	15	24	<0.001**
					100	

****- highly significant at 1 level.**



In our study, a total of thirty nine patients had some degree of systolic LV dysfunction at 48 hours.

In the systolic dysfunction group at 48 hours, 24 patients had an EF of less than 40% and fifteen (62.5%) amongst them belonged to the severe stroke group which is statistically highly significant.

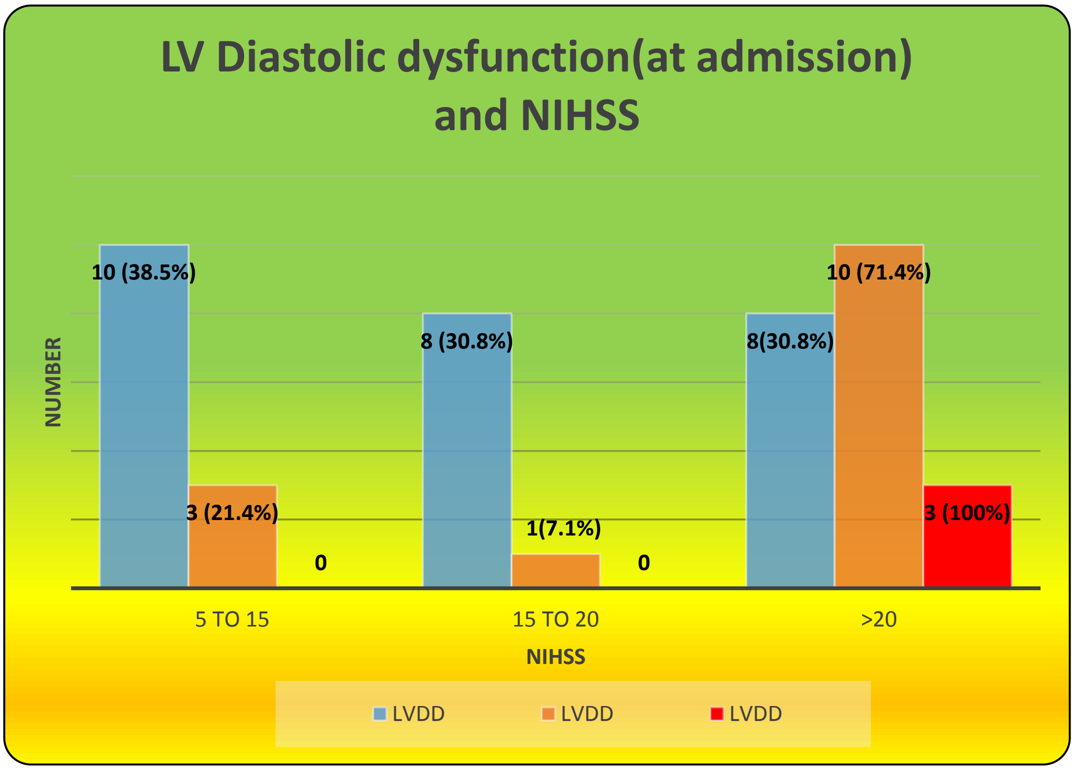
P value obtained was <0.001

Sixty one patients had normal LV systolic function and thirty seven (60.7%) among them belonged to the moderate stroke group and this difference among stroke severity groups is statistically highly significant.

Table: 14 SEVERITY OF LV DIASTOLIC DYSFUNCTION (at admission) AMONG STROKE SEVERITY GROUPS

LVDD Grade		NIHSS				p value
		5-15	16-20	>20	total	
	normal	34	15	8	57	<0.001**
	1	10	8	8	26	
	2	3	1	10	14	
	3	0	0	3	3	
					100	

- highly significant at 1 level.



In our study, a total of forty three patients had some degree of LV diastolic dysfunction during admission.

In the LVDD group at admission, three patients had Grade 3 LVDD and all 3 (100%) of them belonged to the severe stroke group which is statistically highly significant.

P value obtained was <0.001

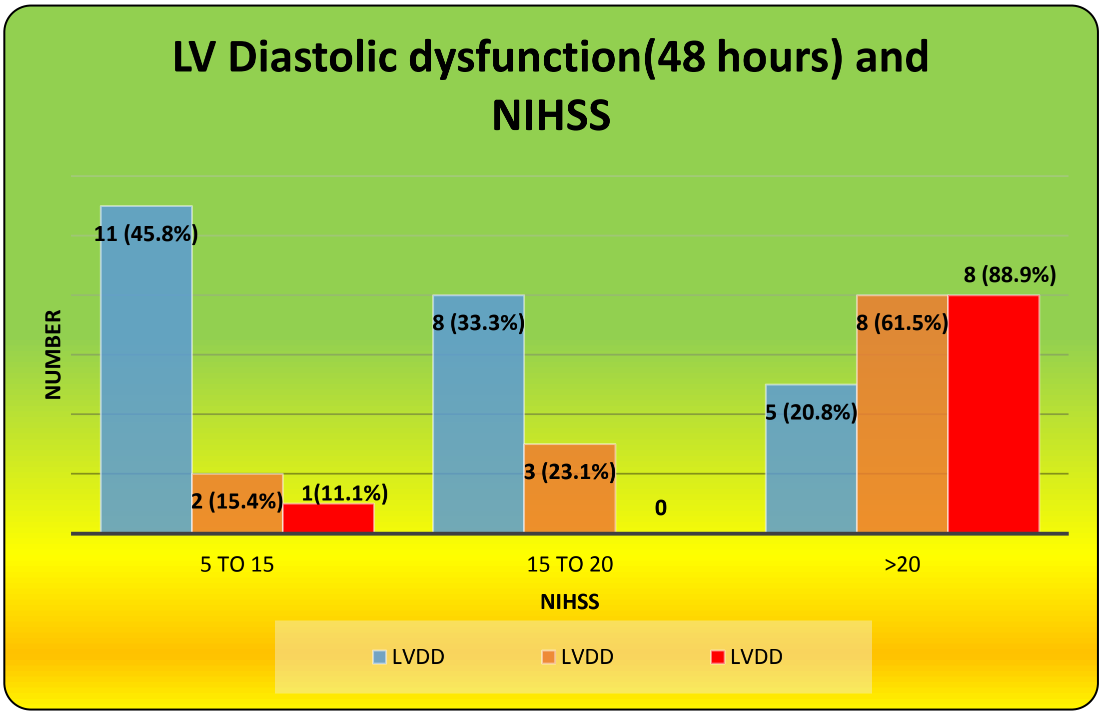
Fourteen patients had Grade2 LVDD and 10 of them (71.4%) belonged to severe stroke group which is statistically highly significant. ($p<0.001$)

Fifty seven patients had normal LV diastolic function and 34 (59.6%) of them belonged to the moderate stroke group and this difference among severity groups is statistically highly significant.

Table: 15 SEVERITY OF LV DIASTOLIC DYSFUNCTION (at 48 hours) AMONG STROKE SEVERITY GROUPS

LVDD Grade		NIHSS				p value
		5-15	16-20	>20	total	
	normal	33	13	8	54	<0.001**
	1	11	8	5	24	
	2	2	3	8	13	
	3	1	0	8	9	
					100	

****- highly significant at 1 level.**



In our study, a total of forty four patients had some degree of LV diastolic dysfunction at 48 hours.

In the LVDD group at 48 hours, nine patients had Grade 3 LVDD and 8 (88.9%) of them belonged to the severe stroke group which is statistically highly significant.

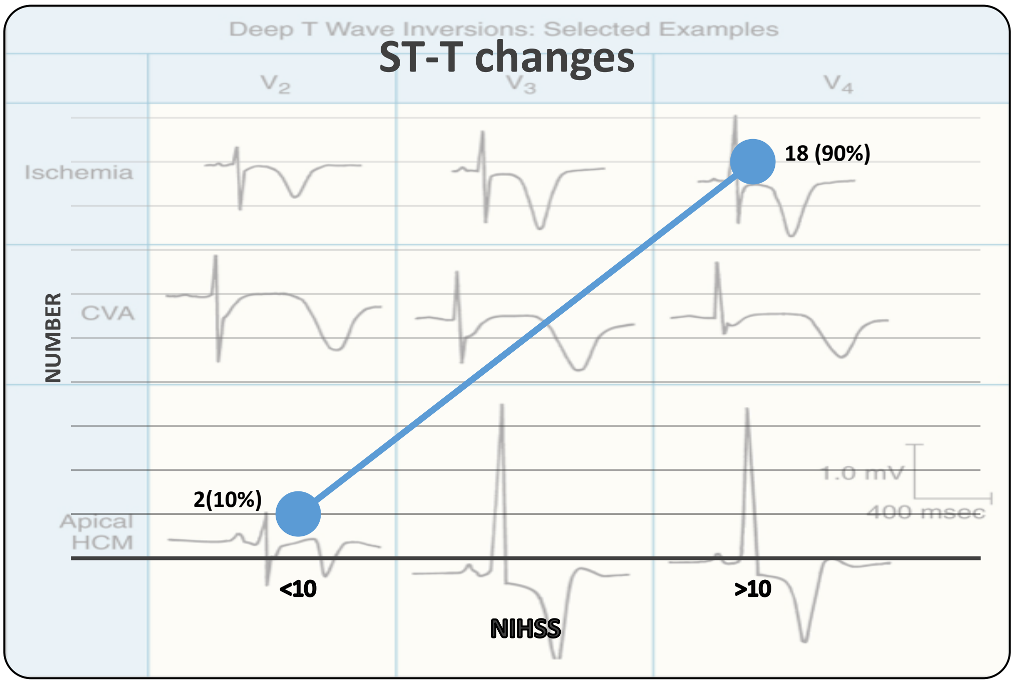
P value obtained was <0.001

Thirteen patients had Grade2 LVDD and 10 of them (61.5%) belonged to severe stroke group which is statistically highly significant. (p<0.001)

Fifty four patients had normal LV diastolic function and 33 (61.1%) of them belonged to the moderate stroke group and this difference among severity groups is statistically highly significant.

Table:16 ST-T CHANGES ACROSS STROKE SEVERITY GROUPS (at median NIHSS 10)

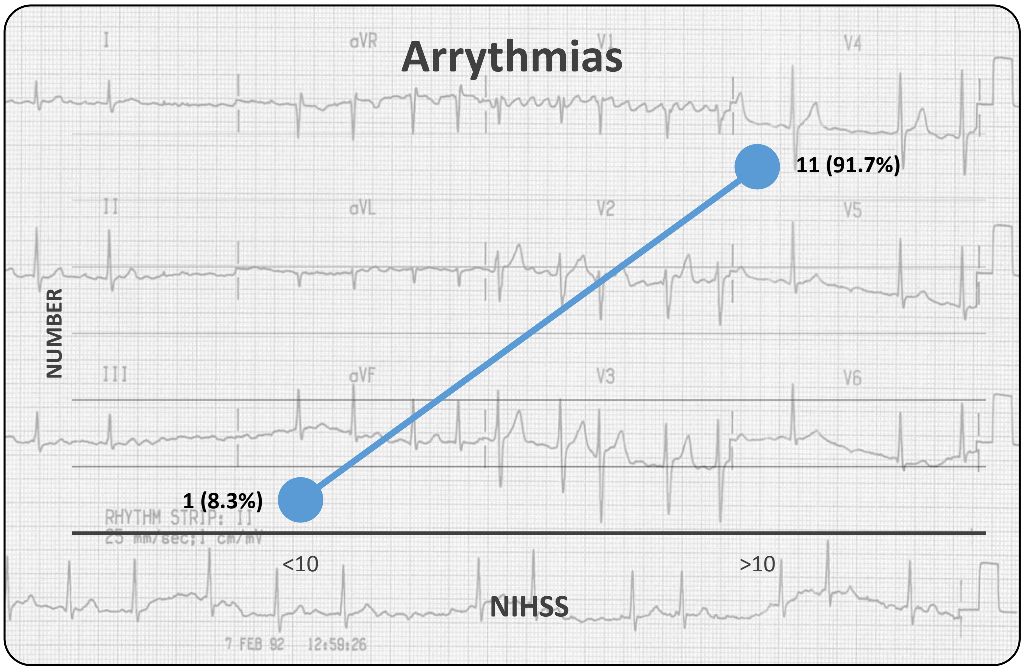
ST-T changes	NIHSS		total
	<10	>10	
	2(10%)	18(90%)	20



In our study, twenty patients had ST-T changes in their ECG at the end of 48 hours and among them 18 (90%) patients belonged to the NIHSS >10 group.

TABLE:17 ARRHYTHMIAS DISTRIBUTED ACROSS STROKE SEVERITY (at median NIHSS 10)

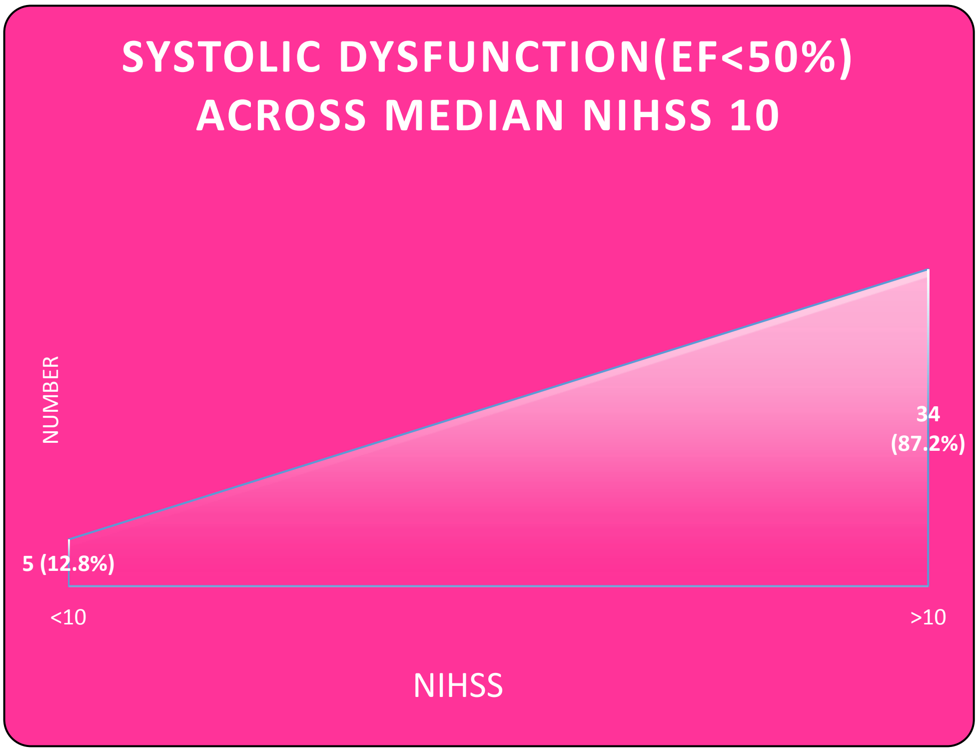
Arrythmias	NIHSS		total
	<10	>10	
	1(8.3%)	11(91.7%)	12



In our study, twelve patients had ST-T changes in their ECG at the end of 48 hours and among them 11 (91.7%) patients belonged to the NIHSS >10 group.

TABLE:18 ARRHYTHMIAS DISTRIBUTED ACROSS STROKE SEVERITY (at median NIHSS 10)

	NIHSS		total
	<10	>10	
EF<50%	5(87.2%)	34(12.8%)	39

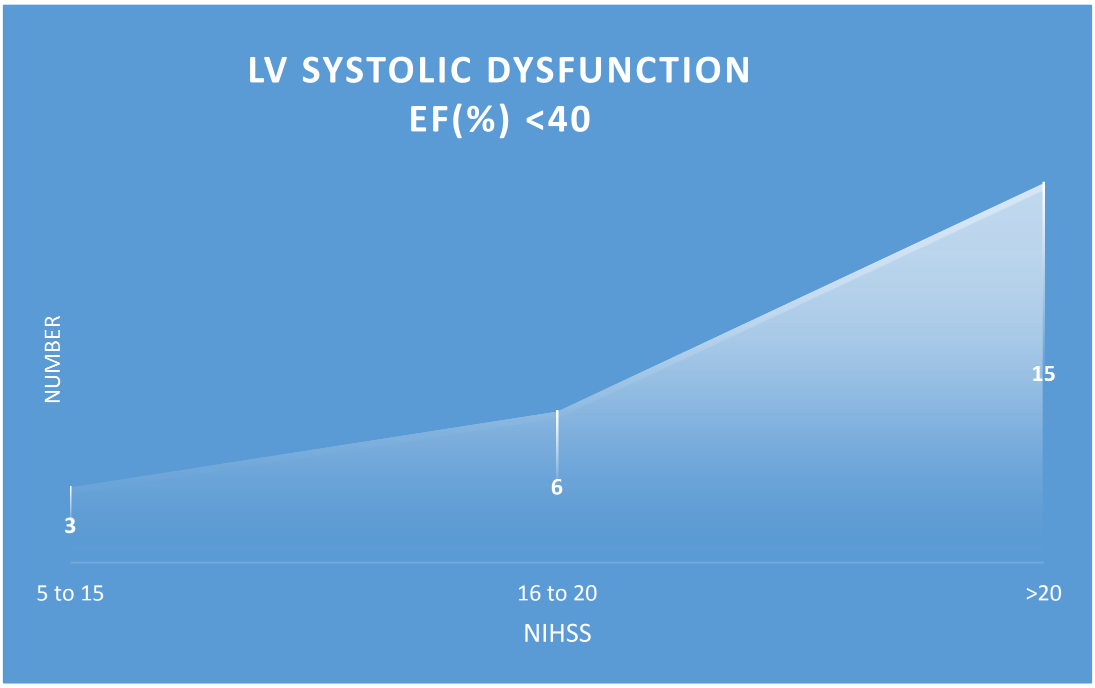


In our study, 39 patients had some degree of LVSD in the echo at the end of 48 hours and among them 34 (87.2%) patients belonged to the NIHSS >10 group.

Table: 19 DISTRIBUTION OF MODERATE TO SEVERE LVSD ACROSS STROKE SEVERITY GROUPS

		EF<40%		p-value
		frequency	**percentage**	
NIHSS	5 - 15	3	12.5	0.008**
	16 - 20	6	25.0	
	>20	15	62.5	
		24	100.0	

****- highly significant at 1 level**

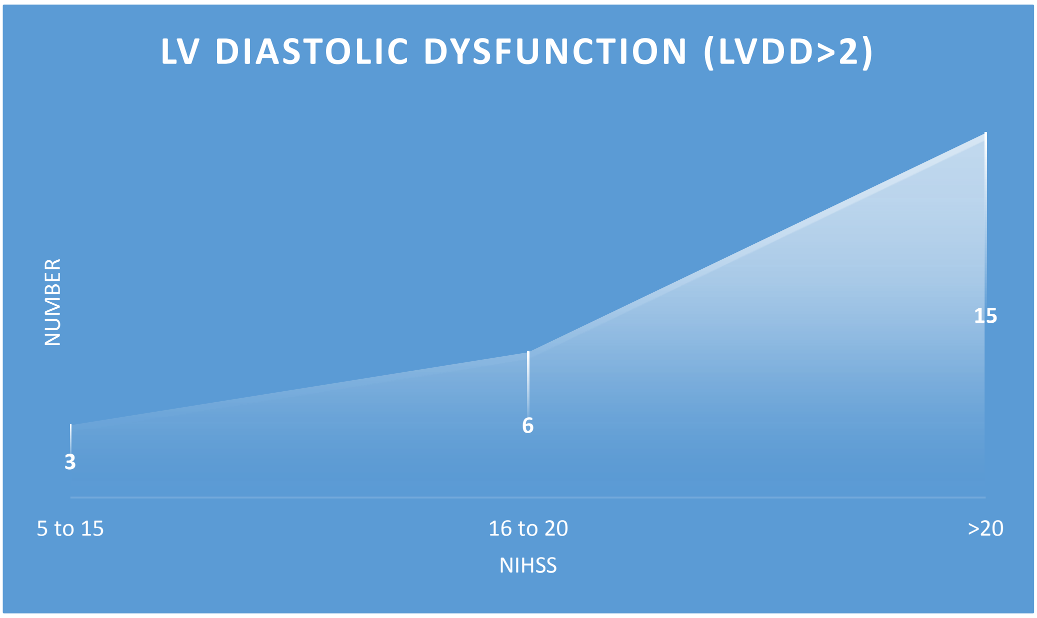


This graph shows the rising trend of the moderate to severe LVSD curve across the stroke severity group. 15 patients(62.5%) fall in the severe stroke group. The difference in distribution is statistically highly significant. (p<0.001)

Table: 20 DISTRIBUTION OF GRADE 2-3 LVDD ACROSS STROKE SEVERITY GROUPS

NIHSS		LVDD Grade 2-3		p-value
		frequency	percentage	
	5 - 15	1	11.1	0.020*
	16 - 20	0	0	
	>20	8	88.9	
		9	100.0	

***- significant at 5 level**

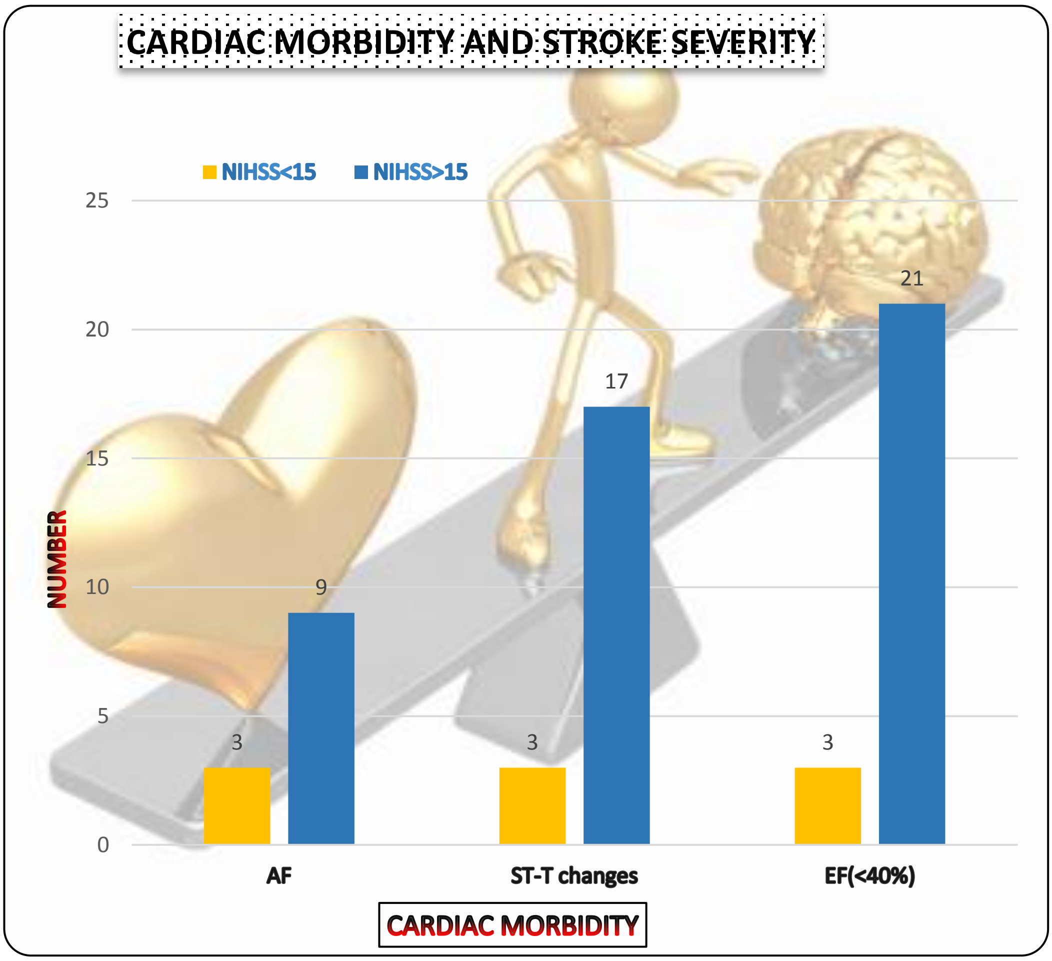


This graph shows the rising trend of the Grade2-3 LVDD curve across the stroke severity group. 8 patients (88.9%) fall in the severe stroke group. The difference in distribution is statistically significant. (p<0.050)

Table :21 SUMMARY OF CARDIAC MORBIDITY ACROSS STROKE SEVERITY

NIHSS		ARRHYTHMIAS	ST-T	EF<40%	p-value
	<15	3	3	3	<0.05*
	>15	9(81.81%)	17(85%)	21(87.5%)	
	total	11	20	24	

*- significant at 5 level



The graph above summarizes the incidence of cardiac complications in acute ischemic stroke.

The number of patients with AF, ST-T changes and moderate to severe LV dysfunction are divided with an arbitrary median of NHISS 15.

81.1% of AF, 85% of ST-T changes, 87.5% of significant LV systolic dysfunction were found in the 'moderate to severe' and 'severe' stroke groups (NIHSS>15).

RESULTS

- The total sample size of our study is 100. All the patients selected met the inclusion and exclusion criteria.

DEMOGRAPHICS

- Demographic data of our study group is summarized in table 1 and table 2.
- Age group distribution followed a bell shaped curve with majority (65%) of the population in 50 to 70 years of age. NIHSS severity among the age groups were more in the same age group.
- 6(20.2%) in the age group 51-60 and 14(48.2%) in the age group 61-70 belonged to NIHSS>20 group
- 54% were males and 46% females. Study population had a fair sex ratio.

RISK FACTORS

- Of the 100 patients, 57 had hypertension, 31 had diabetes mellitus, 20 had hyperlipidemia.
- 13 consumed alcohol, 12 had a history of CAD, 9 suffered heart failure and 5 had CKD

- Thyroid disease was seen in 4 patients and 2 patients had previous CVA.

ARRYTHMIAS

- 7 patients had arrhythmias detected during admission and 12 patients had arrhythmias at the end of 48 hours
- Of the 12 patients, 11 had Atrial fibrillation and 1 developed ventricular tachycardia
- 11 (91.7%) of those 12 patients who had arrhythmias also had an NIHSS>10

ST-T CHANGES

- 14% had ST-T changes in the ECG at admission. 20% had ST-T changes in the ECG at 48 hours.
- ST-T changes included onlyischemic changes excluding non-specific ST-T changes andST depression &T wave inversions seen in acute strokes.
- 18 (90%) of those 20 patients who had ST-T changes had an NIHSS>10

LV SYSTOLIC DYSFUNCTION

- Systolic dysfunction was assessed based on Ejection fraction.

- 12 patients had EF<40% at admission, and the number in this group increased to 24 at 48 hours
- Patients with EF 41-50% at admission and 48 hours were 23 and 15 respectively
- Patients with EF>60 at admission and 48 hours were 65 and 61
- In the systolic dysfunction group at admission, twelve patients had an EF of less than 40% and ten (83.3%) amongst them belonged to the severe stroke group which was statistically highly significant.(p<0.001)
- In the systolic dysfunction group at 48 hours, 24 patients had an EF of less than 40% and fifteen (62.5%) amongst them belonged to the severe stroke group which is statistically highly significant.(p<0.001)
- 24 patients had an EF<40 at 48hours and 15 (62.5%) of them had a NIHSS>20 and 21 (87.5%) of them had a NIHSS>15, which is highly statistically significant. (p<0.010)

LV DIASTOLIC DYSFUNCTION

- 17 patients had LVDD Gr.2 and 3 at admission , and the number in this group increased to 21 at 48 hours

- Patients with LVDD Gr1 at admission and 48 hours were 26 and 24 respectively.
- Patients with normal diastolic function at admission and 48 hours were 57 and 54. The seeming reduction in number is due to shift to severe LVDD groups.
- In the LVDD group at admission, three patients had Grade 3 LVDD and all 3 (100%) of them belonged to the severe stroke group which is statistically highly significant ($p<0.001$)
- In the LVDD group at 48 hours, nine patients had Grade 3 LVDD and 8 (88.9%) of them belonged to the severe stroke group which is statistically highly significant. ($p<0.001$)
- In total, 9 patients had an LVDD Gr2 and 3 and 8 (88.9%) of them had a NIHSS>20 which is statistically significant. ($p<0.050$)

DISCUSSION

There had been a constant emphasis on the complex relationship between cerebrovascular accidents and cardiovascular diseases. Many studies had been made in this context. Yet, the studies varied in many aspects and laid importance on one aspect out of the many complex manifestations involved with cardiovascular and cerebrovascular systems.

A number of studies in the past have concentrated upon the varied ECG manifestations occurring in acute CVA including both ischemic and hemorrhagic. But whether similar effects cause significant damage in ischemic/thrombotic stroke has been less studied. The insular cortex had been studied and found to cause cardiac sympathetic neural upregulation and ECG abnormalities.

A number of neurosurgical studies have shown that ECG abnormalities and left ventricular dysfunction (wall motion hypokinesias) can occur in hemorrhagic stroke especially SAH.[40-43] Also described are myocardial stunning and myocardial necrosis. Other findings were increased levels of natriuretic factors, catecholamines in the plasma. Myocardial perfusion too gets affected regionally.[42-43]

When an acute ischemic/thrombotic stroke happens in any patient with underlying heart disease, the damage is severe.[44-45] The autoregulation of blood flow is lost in the ischemic penumbra as the main factor which determines it, ‘the cardiac function’ is under stake.[46]

In our study, the results are similar to that available in the literature, which suggests that the incidence of systolic dysfunction in acute ischemic/thrombotic stroke patients can range from 14% to 30%.

ST-T CHANGES

Some studies also throw light on the ischemic changes in ECG which claims a range of 36% to 74% in hemorrhagic strokes. ‘Oppenheimer et al’ in their study observed an incidence of 15-20% after ischemic stroke. In our study, 20 patients had ischemic changes in the ECG. The incidence goes in hand with the literature. And also 18 out of 20 had a NIHSS>10

These changes occur due to a neural mechanism and not associated CAD.

SYSTOLIC DYSFUNCTION

24 patients had an EF<40 at 48hours and 15 (62.5%) of them had a NIHSS>20 and 21 (87.5%) of them had a NIHSS>15, which is highly statistically significant. ($p<0.010$)

The incidence of 24% found in our study is similar to that quoted in other studies in this domain.

A similar study by 'Wira et al' observed a systolic dysfunction of 28.5%

In our study, the study design did not include a control group nor was echocardiography repeated in the follow up period in all the patients. Hence, whether the systolic dysfunction is a transient one or a pre-existing one could not be ascertained. Yet from the demographics of our study, it is found that only 9 patients had preexisting heart failure. So, the possibility of a temporal association of the systolic dysfunction and acute ischemic stroke is definitely feasible.[50-52] Nevertheless, from our study we found that systolic dysfunction correlated well with the NIHSS severity as similar correlations with mortality were observed in other studies.[54]

ARRHYTHMIAS

11 patients in our study had atrial fibrillation. One patient had a VT. About atrial fibrillation there only one study[53] which proposes AF as a predictor of mortality. In our study 11 out of 12 patients had an NIHSS>10 which is an indirect predictor of mortality. The exact mechanisms of how mortality is increased is poorly understood.

An NIHSS of 10 is chosen as a comparing destination point because few studies in the literature have found that the excellent prognosis rate decreases significantly when the NIHSS is more than 10.

All these changes are associated with increased mortality which was reflected in our study by the severity of NIHSS. Death was not taken as an

outcome in our study.

A study of stroke patients had shown that left insular cortical lesions are more likely to produce ECG abnormalities and cardiac sympathetic neural upregulation. Such changes can lead to increased cardiac mortality which can be a major cause of mortality in stroke.[47-49]

LIMITATIONS

Our study is limited by its sample size which is small.

The study design did not include a control group nor was echocardiography repeated in the follow up period in all the patients. Hence, whether the systolic dysfunction is a transient one or a pre-existing one could not be ascertained.

Higher rate of systolic dysfunction were not compared with age matched controls. Yet from the demographics of our study, it is found that only 9 patients had preexisting heart failure. So, the possibility of a temporal association of the systolic dysfunction and acute ischemic stroke is definitely feasible.

Nevertheless, from our study we found that systolic dysfunction correlated well with the NIHSS severity as similar correlations with mortality were observed in other studies. Yet, this is a considerable finding warranting further studies.

Another limitation is that, in our study we did not control for other factors affecting LV function and any other previous medications which would affect the LV function during the time of performing the echocardiography.

CONCLUSION

- A subset of patients who suffer acute ischemic/thrombotic stroke develop an array of cardiac complications.
- These cardiac manifestations are either caused directly by the neural effects or other reasons not within the scope of this study.
- Active atrial fibrillation, ischemic changes in the ECG, Left ventricular systolic and diastolic dysfunction are associated more with moderate to severe and severe stroke groups.
- Thus these subset of patients may be associated with higher in hospital mortality rates and poor outcomes.
- This study emphasizes the role of cardiac monitoring in the acute stroke setting.
- Further studies can throw light on cardiac augmentation strategies, which could be adopted in the management protocol of acute ischemic/thrombotic stroke patients.

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PROFORMA

Study: CARDIAC COMPLICATIONS IN ACUTE ISCHEMIC/ THROMBOTIC STROKE WITH SPECIAL REFERENCE TO VENTRICULAR DYSFUNCTION.

Name: Patient ID No. :

Age/Sex:

IP No.:

Risk factors	
☐ Diabetes mellitus	☐ Hyperlipidemia
☐ Hypertension	☐ CKD
☐ Heart failure	☐ Thyroid disorder
☐ Alcoholism	☐ Previous CVA/ TIA

Clinical parameters		
	At admission	At 48 hours
Pulse		
BP		

CNS examination:

Investigations:

CBC		RFT			LFT		
Hb		Glucose		mg/dl	T.Bili		mg/dl
Hct		Urea		mg/dl	D.Bili		mg/dl
TC		Creat.		mg/dl	SGOT		U/l
DC-N		Na^+		mEq/l	SGPT		U/l
-L		K^+		mEq/l	ALP		U/l
-E					T.Protein		g/dl
Platelet					Albumin		g/dl

CT brain(plain):

Echocardiography		
	At admission	At 48 hours
EF%		
LVDD		

ECG		
	At admission	At 48 hours
Ischemic changes		
Arrhythmias		

NIH Stroke scale:

	Neurological examination	Score
1a.	Level of consciousness	
1b.	LOC questions	
1c.	LOC commands	
2.	Best gaze	
3.	Visual	
4.	Facial palsy	
5a.	Motor arm (L)	
5b.	Motor arm (R)	
6a.	Motor leg (L)	
6b.	Motor leg (R)	
7.	Limb ataxia	
8.	Sensory	
9.	Best language	
10.	Dysarthria	
11.	Extinction and Inattention	
	TOTAL SCORE	

INFORMATION SHEET

Study Title	:	**CARDIAC COMPLICATIONS IN ACUTE ISCHEMIC / THROMBOTIC STROKE PATIENTS WITH SPECIAL REFERENCE TO VENTRICULAR DYSFUNCTION**
Study Centre	:	**Rajiv Gandhi Government General Hospital, Chennai.**
Patient's Name/Age	:	
Investigators Name	:	
Identification Number	:	

- We are conducting a study on "**CARDIAC COMPLICATIONS IN ACUTE ISCHEMIC / THROMBOTIC STROKE PATIENTS WITH SPECIAL REFERENCE TO VENTRICULAR DYSFUNCTION**" among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.
- The purpose of this study is we are selecting certain patients affected with stroke and if you are found eligible, we will perform an ecg and echocardiography(which are non-invasive tests) to assess your cardiac status. Apart from routuine blood examination,we may perform extra tests and special studies which in any way do not affect your final report or management.
- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.
- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator | Signature of Participant

Date :

PATIENT CONSENT FORM

Study Title	:	**CARDIAC COMPLICATIONS IN ACUTE ISCHEMIC / THROMBOTIC STROKE PATIENTS WITH SPECIAL REFERENCE TO VENTRICULAR DYSFUNCTION**
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name/Age	:	
Investigators Name	:	
Identification Number	:	

- I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.
- I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.
- I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.
- I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.
- I hereby consent to participate in this study.
- I hereby give permission to undergo hematological test.

Signature of Investigator   Signature of Participant

Date:

<u>ஆராய்ச்சி தகவல் தாள்</u>

- இராஜீவ் காந்தி அரசு பொது மருத்துவமனைக்கு வரும் **"லையில் இரத்த குழாய் அடைப்பினால் ஏற்படும் பக்கவாதம் உள்ள நோயாளிகளுக்கு உண்டாகும் இருதய பாதிப்புகளை அறிவதே"** இவ்வாராய்ச்சியின் நோக்கமாகும்.

- நீங்கள் இந்த ஆராய்ச்சிக்கு தகுதியானவராய் இருக்குமானால் உங்களுடைய இருதயத்தின் நிலையை இதயத்துடிப்பு பதிவி (ECG) மற்றும் மின் ஒலி இதய வரைவி (Echocardiography) பரிசோதனைகள் லம் அறிவோம்.

- இந்த ஆராய்ச்சியில் வழக்கமாக செய்யும் இரத்த பரிசோதனைகளுடன் சில சிறப்பு இரத்த பரிசோதனைகளை செய்து அதன் தகவல்களை அறிவோம்.

- நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்புக்கு உட்படாது என்பதையும் தெரிவித்துக் கொள்கிறோம்.

- முடிவுகளை அல்லது கருத்துக்களை வெளியிடும்போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

- இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின் வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

- இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி :

<u>ஆராய்ச்சி ஒப்புதல் கடிதம்</u>

<u>ஆராய்ச்சி தலைப்பு:</u>

இரத்த குழாய் அடைப்பினால் ஏற்படும் பக்கவாதம் உள்ள நோயாளிகளுக்கு உண்டாகும் இருதய பாதிப்புகளை அறிவதே இவ்வாராய்ச்சியின் நோக்கமாகும்.

பெயர் : தேதி :

வயது : உள் நோயாளி எண் :

பால் : ஆராய்ச்சி சேர்க்கை எண் :

- இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு முழுவதும் தெளிவாக விளக்கப்பட்டது.
- எனக்கு விளக்கப்பட்ட விஷயங்களை புரிந்துக் கொண்டு நான் என் சம்மதத்தைத் தெரிவிக்கிறேன்.
- எனக்கு இரத்தப் பரிசோதனை செய்து கொள்ள சம்மதம்.
- இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து என்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் புரிந்து கொண்டேன்.
- நான் இரத்தகுழாய் அடைப்பினால் ஏற்படும் பக்கவாதம் நோய் குறித்து இந்த ஆராய்ச்சியின் விபரங்களைக் கொண்ட தகவல்தாளைப் பெற்றுக் கொண்டேன்.
- இதன் □லம் எந்த பின் விளைவும் வராது என மருத்துவர் □லம் புரிந்து கொண்டு என்னுடைய சுய நினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள சம்மதிக்கிறேன்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி:

ID no.	Age	Sex	ECG				Echocardiography				NIHSS	Risk factors								
			arrhythmias		ST-T changes		EF %		LVDD(Gr.)			DM	HTN	CAD	HF	Alc	HL	CKD	Thy	old CVA
			adm	48h	adm	48h	adm	48h	adm	48h										
1	56	m	n	n	n	n	60	55	0	0	8	y	n	n	n	n	n	n	n	n
2	66	m	n	n	n	n	62	62	1	1	15	n	y	n	n	n	n	n	n	n
3	74	m	y	y	n	n	60	58	0	0	14	y	y	n	n	n	n	n	n	y
4	55	m	n	n	y	y	44	30	3	3	24	y	y	y	y	n	n	n	n	n
5	55	f	n	n	n	n	60	60	0	0	8	n	n	n	n	n	n	n	y	n
6	57	f	n	n	n	n	58	56	0	0	8	n	y	n	n	n	n	n	n	n
7	60	f	n	n	n	n	44	42	0	0	16	y	y	n	n	n	y	n	n	n
8	64	m	n	n	n	y	62	62	0	0	8	y	n	n	n	y	n	n	n	n
9	80	m	n	y	n	n	54	54	1	1	12	n	y	n	n	n	n	n	n	y
10	51	m	n	n	n	n	55	45	1	1	11	n	n	n	n	n	n	n	n	n
11	69	f	n	n	n	n	36	30	1	3	28	y	y	n	n	n	y	n	n	n
12	58	f	n	n	n	y	48	45	0	0	16	n	y	n	n	n	n	n	n	n
13	70	m	n	n	n	n	60	60	2	2	12	y	n	n	n	n	n	n	n	n
14	49	m	n	n	n	n	62	62	0	0	12	y	n	n	n	y	n	n	n	n
15	64	f	y	y	n	n	46	40	0	0	24	y	y	y	n	n	n	n	n	y
16	58	m	n	n	n	n	65	60	0	0	9	n	n	n	n	n	n	n	n	n
17	44	m	n	n	n	n	58	55	0	0	14	n	y	n	n	n	n	n	y	n
18	56	m	n	n	n	n	38	38	2	3	32	n	y	n	n	n	n	n	n	y
19	77	f	n	n	y	y	44	38	0	0	16	y	y	n	y	n	n	n	n	n
20	63	f	n	n	n	n	66	64	0	0	8	n	n	n	n	n	y	n	n	n
21	54	f	n	n	n	n	52	52	0	0	16	n	y	n	n	n	y	n	n	n
22	75	m	n	n	n	n	55	50	1	1	11	n	n	n	n	n	n	n	n	n
23	66	m	n	n	n	n	62	62	0	0	8	n	y	y	n	n	n	n	n	n

24	51	m	n	n	n	n	64	64	0	0	14	y	n	n	n	n	n	n	n	n
25	79	f	n	n	y	y	56	55	1	1	24	y	y	n	n	n	n	n	n	y
26	62	m	n	n	n	n	66	64	1	1	16	n	n	n	n	y	y	n	n	n
27	55	f	n	n	n	n	46	46	2	2	14	n	n	n	n	n	n	n	n	n
28	69	m	n	n	n	n	55	55	2	2	28	y	y	y	n	n	n	y	n	y
29	49	f	n	n	n	n	62	62	0	0	8	n	n	n	n	n	n	n	n	n
30	56	f	n	n	n	n	54	54	0	0	14	n	n	n	n	n	n	n	n	n
31	81	m	n	n	y	y	45	30	2	2	20	y	n	n	n	n	y	y	n	y
32	78	m	n	y	n	n	56	56	0	0	24	y	y	n	n	n	n	n	n	y
33	74	f	n	n	n	n	64	60	0	0	16	n	n	y	n	n	n	n	n	n
34	60	f	n	n	n	n	40	36	1	1	8	n	y	n	n	n	n	n	n	n
35	70	m	n	n	n	y	64	64	0	0	14	n	y	n	n	n	n	n	n	n
36	71	f	y	y	n	n	54	54	0	0	16	n	y	y	n	n	n	n	n	n
37	45	f	n	n	n	n	58	56	0	0	12	n	n	n	n	n	y	n	n	n
38	52	f	n	n	n	n	44	36	1	2	25	y	y	n	n	n	n	n	n	n
39	49	m	n	n	n	n	60	56	1	1	8	n	n	n	n	y	n	n	n	n
40	55	f	n	n	n	n	62	56	0	0	16	n	y	n	n	n	n	n	n	n
41	60	m	n	n	n	y	36	36	2	2	32	n	y	n	n	n	n	n	n	y
42	48	f	n	n	n	n	60	60	0	0	12	y	n	n	n	n	n	n	n	n
43	46	m	n	n	n	n	64	58	1	1	16	n	y	n	n	n	y	n	n	n
44	53	f	n	n	n	n	64	64	0	0	12	n	n	n	n	n	n	n	n	n
45	57	f	n	n	y	y	54	54	1	1	26	y	y	n	n	n	n	y	n	n
46	60	m	n	n	n	n	40	38	1	1	20	y	n	y	y	n	n	y	n	n
47	63	m	n	y	n	n	56	55	0	0	18	n	n	n	n	n	y	n	n	y
48	53	f	n	n	n	n	62	60	0	0	8	n	n	n	n	n	n	n	n	n
49	77	f	n	n	n	n	56	56	0	0	12	n	n	n	n	n	n	n	n	n
50	38	m	n	n	n	n	60	55	0	0	12	n	y	y	n	y	n	n	n	n

51	42	m	n	n	n	n	64	62	0	0	8	n	n	n	n	y	n	n	n	n
52	84	m	n	n	y	y	40	35	2	3	30	n	y	n	n	n	n	n	n	n
53	79	f	n	n	n	n	64	62	0	0	10	n	y	n	n	n	y	n	n	n
54	63	m	y	y	n	n	54	52	0	0	20	n	y	n	n	n	n	n	n	n
55	65	f	n	n	y	y	48	48	1	1	7	n	y	n	n	n	n	n	n	n
56	37	m	n	n	n	n	66	64	0	0	18	y	n	n	n	y	y	n	n	n
57	45	m	n	n	n	n	54	54	0	0	12	n	n	n	n	y	n	n	n	y
58	50	m	n	n	n	n	64	62	1	1	14	n	y	n	n	n	n	n	n	n
59	53	f	n	n	n	n	60	60	0	0	10	n	n	n	n	n	n	n	y	n
60	68	m	n	n	y	y	35	28	2	2	36	n	y	y	y	n	n	n	n	y
61	62	f	n	n	n	n	46	44	1	1	24	n	y	n	n	n	n	n	n	n
62	48	f	n	n	n	n	66	66	0	0	12	n	n	n	n	n	y	n	n	n
63	53	m	n	n	n	n	56	54	0	0	28	n	y	n	n	y	n	n	n	n
64	67	m	n	n	n	n	44	44	0	0	30	n	y	n	n	n	n	n	y	n
65	73	f	n	n	y	y	56	56	1	1	28	y	y	n	n	n	n	n	n	y
66	76	m	n	n	n	n	48	44	0	0	18	n	y	n	n	n	n	n	n	y
67	59	m	n	n	n	n	45	40	0	1	8	n	n	n	n	n	n	n	n	n
68	78	m	n	n	y	y	55	55	0	0	16	n	n	y	n	n	n	n	n	n
69	66	f	n	y	n	n	50	44	0	0	24	n	y	n	n	n	n	n	n	n
70	43	f	n	n	n	n	55	40	0	1	18	y	n	n	n	n	n	n	n	n
71	37	m	n	n	n	n	66	66	0	0	12	n	n	n	n	y	n	n	n	n
72	50	m	n	n	n	n	62	52	0	0	14	n	y	n	n	n	y	n	n	n
73	66	f	n	n	n	n	30	30	2	2	22	n	y	y	y	n	n	n	n	n
74	84	f	y	y	n	n	44	38	1	1	10	n	n	n	y	n	n	n	n	n
75	75	f	n	n	n	n	54	54	0	0	22	y	y	n	n	n	n	n	n	y
76	81	m	n	n	y	y	38	30	2	3	28	n	y	n	n	n	n	n	n	y
77	65	f	n	n	n	n	56	52	0	0	12	n	n	n	n	n	y	n	n	n

78	77	f	n	n	n	n	50	50	1	1	24	y	y	n	n	n	n	n	n	n
79	70	m	n	n	y	y	34	30	3	3	30	n	y	n	n	n	y	n	n	n
80	68	m	y	y	n	n	55	55	0	0	26	y	y	n	n	n	n	n	n	n
81	58	f	n	n	n	n	60	60	0	0	8	n	y	n	n	n	n	n	n	n
82	73	f	n	n	n	n	62	60	1	1	16	n	n	n	n	n	y	n	n	y
83	84	m	n	n	n	n	42	36	1	2	26	y	y	n	y	n	n	y	n	n
84	55	f	n	n	n	n	54	50	0	0	14	n	y	n	n	n	n	n	n	n
85	70	f	n	n	y	y	60	54	2	2	27	n	y	n	n	n	y	n	n	n
86	86	m	n	n	n	n	48	44	0	0	14	n	n	y	n	y	n	n	n	y
87	43	m	n	n	n	n	65	65	0	0	8	y	n	n	n	y	y	n	n	n
88	72	m	n	n	n	y	54	54	1	1	18	n	n	n	n	n	n	n	n	n
89	64	f	n	n	y	y	40	34	2	3	22	n	y	n	n	n	n	n	n	n
90	47	f	n	n	n	n	64	58	0	0	12	y	n	n	n	n	y	n	n	n
91	59	m	n	y	n	n	54	54	1	2	20	n	y	n	n	n	n	n	n	n
92	49	m	n	n	n	n	42	40	1	1	16	n	y	n	n	y	n	n	n	n
93	75	m	n	n	n	n	38	36	3	3	34	y	y	n	n	n	n	n	n	y
94	61	f	y	y	n	n	52	52	1	2	16	n	y	n	n	n	n	n	n	n
95	67	m	n	n	n	n	42	28	2	2	30	y	y	n	y	n	n	n	n	n
96	65	m	n	n	n	n	64	64	0	0	22	n	y	n	n	n	n	n	n	n
97	84	f	n	n	n	n	50	46	2	3	8	n	y	n	n	n	n	n	n	n
98	48	m	n	n	n	n	66	62	1	1	10	n	n	n	n	n	y	n	n	n
99	57	f	n	n	n	n	46	44	0	0	16	y	n	n	n	n	n	n	n	n
100	62	m	n	n	n	y	42	36	0	1	16	n	n	n	y	n	n	n	n	y

KEY TO MASTER CHART

m	-	Male
f	-	Female
y	-	Yes / present
n	-	No / absent
ECG	-	Electrocardiogram
EF	-	Ejectionfraction
LVDD	-	Left Ventricular Diastolic Dysfunction
NIHSS	-	National Institute of Health Stroke Scale
CAD	-	Coronary artery disease
CKD	-	Chronic kidney disease
HL	-	Hyperlipidemia
HTN	-	Hypertension
DM	-	Diabetes mellitus
HF	-	Heart failure
Alc	-	Alcohol intake
Thy	-	Thyroid disorders
CVA	-	Cerebrovascular accident

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. Vivek .M,
Post Graduate, MD (General Medicine)
Institute of Internal Medicine,
Madras Medical College,
Chennai – 600003.

Dear Dr. Vivek .M,

The Institutional Ethics Committee has considered your request and approved your study titled **"Cardiac complications in Acute Ischemic / Thrombotic Stroke patients with special reference to Ventricular Dysfunction"** No. 28072014.

The following members of Ethics Committee were present in the meeting held on 01.07.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Dr. R. Vimala, M.D., Dean, MMC, Ch-3.	-- Deputy Chair Person
3.	Prof. Kalaiselvi, MD., Vice-Principal, MMC, Ch-3	-- Member Secretary
4.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
5.	Dr. G. Muralidharan, Director Incharge , Inst. of Surgery	-- Member
6.	Prof. Md Ali, MD., DM., Prof & HOD of MGE, MMC, Ch-3.	-- Member
7.	Prof. Ramadevi, Director i/c, Biochemistry, MMC,Ch-3.	-- Member
8.	Prof. Saraswathy, MD., Director, Pathology, MMC, Ch-3.	-- Member
9.	Prof. Tito, Director, i/c. Inst. of Internal Medicine, MMC	-- Member
10.	Thiru. Rameshkumar, Administrative Officer	-- Lay Person
11.	Thiru. S. Govindasamy, BABL, High Court, Chennai-1.	-- Lawyer
12.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
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INTRODUCTION

Any acute insult to the central nervous system has been known to cause a wide array of manifestations in the cardiovascular system. This can include asymptomatic ST-T changes, fatal or non-fatal arrhythmias, ventricular dysfunction, or cardiac dysautonomias.[1-3]

Increase in the levels of serum catecholamines following a stroke[4-6], has been thought to play a role but the intricate mechanisms involved is still an enigma. The intrinsic auto regulation of blood flow is impaired in the ischemic penumbra, making the cerebral perfusion mainly dependent on cardiac function.[7-9]Hence, cardiac dysfunction can lead to detrimental effects in acute stroke patients.

This phenomenon has been well studied in SAH by earlier investigators. But whether similar effects cause significant damage in ischemic/thrombotic stroke has been less studied.[10] Inpatients of acute ischemic stroke undergo echocardiograms to look for a cardio embolic source, but there are no recommendations

1

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INTRODUCTION

Any acute insult to the central nervous system has been known to cause a wide array of manifestations in the cardiovascular system. This can include asymptomatic ST-T changes, fatal or non-fatal arrhythmias, ventricular dysfunction, or cardiac dysautonomias.[1-3]

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